DISSERTATION

ON

A STUDY OF CLINICAL PROFILE OF ACUTE KIDNEY INJURY

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the regulations

for the award of the degree of

M.D. -GENERAL MEDICINE- BRANCH – I

THANJAVUR MEDICAL COLLEGE,

THANJAVUR - 613 004.

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI - 600 032.

APRIL - 2014

CERTIFICATE

This is to certify that this dissertation entitled "**A STUDY OF CLINICAL PROFILE OF ACUTE KIDNEY INJURY."** is the bonafide original work of **Dr.SENTHIL KUMAR.K** in partial fulfilment of the requirements for M.D Branch -I (General Medicine) Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in APRIL - 2014. The period of study was from April - 2013 to October - 2013.

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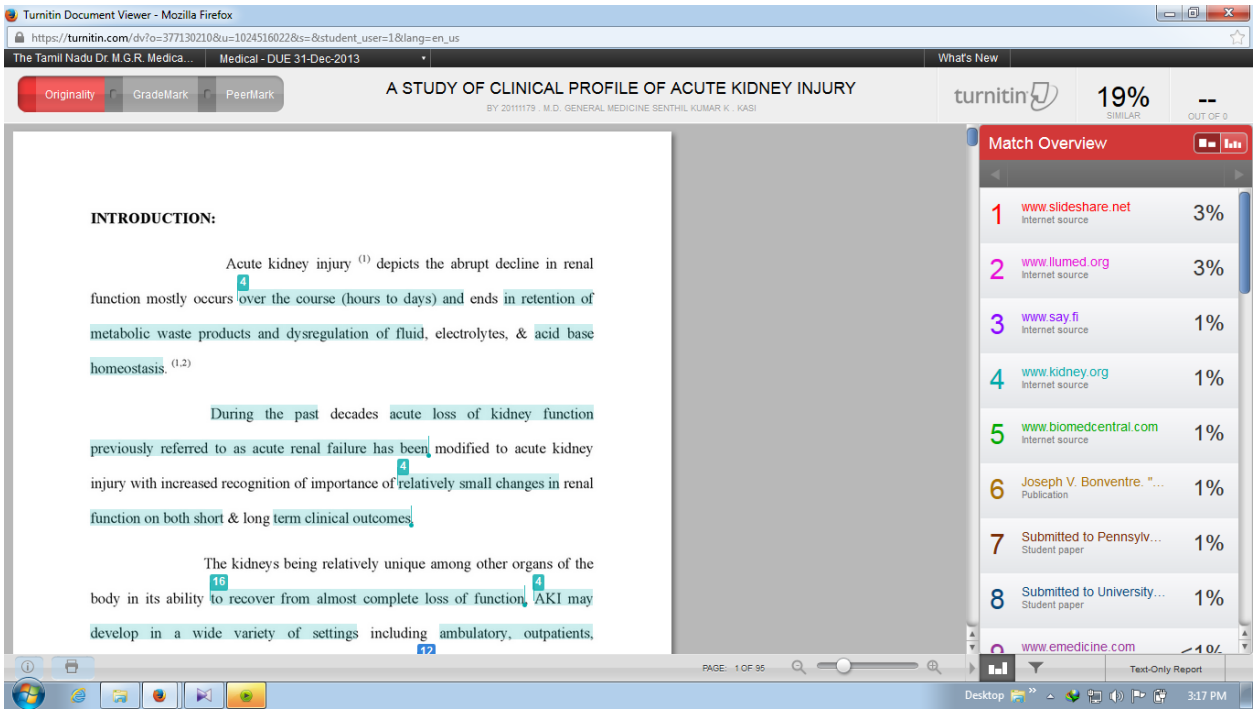
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INTRODUCTION:
Acute kidney injury (1) depicts the abrupt decline in renal function mostly occurs over the course (hours to days) and ends in retention of metabolic waste products and dysregulation of fluid, electrolytes, & acid base homeostasis. (1,2)
During the past decades acute loss of kidney function previously referred to as acute renal failure has been modified to acute kidney injury with increased recognition of importance of relatively small changes in renal function on both short & long term clinical outcomes.
The kidneys being relatively unique among other organs of the body in its ability to recover from almost complete loss of function, AKI may develop in a wide variety of settings including ambulatory, outpatients,
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DECLARATION

I, **Dr. SENTHIL KUMAR.K**, solemnly declare that the dissertation titled **"DISSERTATION ON A STUDY OF CLINICAL PROFILE OF ACUTE KIDNEY INJURY"** is a bonafide workdone by me at Thanjavur Medical College, Thanjavur during april 2013 to October 2013 under the guidance and supervision of **Prof.Dr.C.GANESAN, M.D.,** Unit Chief M-5, Thanjavur Medical College, Thanjavur.

This dissertation is submitted to Tamilnadu Dr. M.G.R Medical University towards partial fulfilment of requirement for the award of **M.D. degree (Branch -I) in General Medicine**.

Place: Thanjavur.

Date: - 12 - 2013. **(Dr.SENTHIL KUMAR.K)**

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CONTENTS

ABBREVATIONS

AKI	–	Acute Kidney Injury
CKD	–	Chronic Kidney Disease
ESRD	–	End Stage Renal Disease
ICU	–	Intensive Care Unit
RRT	–	Renal Replacement Therapy
ADQI	–	Acute Dialysis Quality Initiative
GFR	–	Glomerular Filtration Rate
ARF	–	Acute Renal Failure
ACE	–	Angiotensin Converting Enzyme
NSAID	–	Non Steroidal Anti Inflammatory Drugs
FeNa	–	Fractional Excretion Of Sodium
ATN	–	Acute Tubular Necrosis
ANA	–	Anti Neutrophilic Antibody
ANCA	–	Anti Neutrophilic Cytoplasmic Antibody
AGE	–	Acute Gastroenteritis
LDH	–	Lactate Dehydrogenase
BUN	–	Blood Urea Nitrogen

ABSTRACT

Background: Although the term acute renal failure was replaced by acute kidney injury (AKI) recently, there is a paucity of data on the incidence and profile of AKI in hospitalized patienst from the developing world.

AIMS AND OBJECTIVES: The objective of this study is to determine the etiology, short term outcome and predictors of fatality in patients admitted to thanjavur medical college hospital with AKI, aged more than 12 years.

Materials and Methods: In this prospective observational study, from April 2013 to October 2013, 100 patients admitted with AKI, defined according to the AKI Network criteria. The patients with AKI were followed-up until discharge/death. Their clinical and biochemical data were studied.

Results : Among the 100 patients with acute kidney injury 56 male patients and 44 female patients are included in the study. The common symptoms were oliguria , vomiting ,diarrohea.the common etiologies were acute gastroenteritis(28%), snake bite(20%), septicemia(11%) & acute glomerulonephriris (10%).conservative treatment was done in 45 % of patients ,peritoneal dialysis & haemodialysis done in 55 % of patients and overall mortality was 16 %.

Conclusion: In our study , 84% of patients survived with 45 %of patients treated conservatively and improved with normal renal function. 30% of patients underwent peritoneal dialysis and 26% underwent haemodialysis. In our study mortality is 16% among 84% of survived 68% recovers with normal renal function.

INTRODUCTION

Acute kidney injury [1] depicts the abrupt decline in renal function mostly occurs over the course (hours to days) and ends in retention of metabolic waste products and dysregulation of fluid, electrolytes, & acid base homeostasis. [1,2]

During the past decades acute loss of kidney function previously referred to as acute renal failure has been modified to acute kidney injury with increased recognition of importance of relatively small changes in renal function on both short & long term clinical outcomes.

The kidneys being relatively unique among other organs of the body in its ability to recover from almost complete loss of function, AKI may develop in a wide variety of settings including ambulatory, outpatients, hospitalized, & particularly critically ill patients. AKI is associated with substantial morbidity and mortality.[2]

Although recovery of renal function occurs in majority of patients surviving an episode of AKI, many patients remain dialysis dependant or are left with severe renal impairment. More recently it has been recognized that even patients who have complete or near complete recovery of renal function are at increased risk of CKD and that superimposition of AKI on CKD is associated acceleration in the rate of progression to ESRD.

AKI complicates approximately 5-7% of hospital admission & 30% of admission to ICU patients. The risk of AKI is contributed by the acute insult and background morbidity. Acute insult may be in the forms of sepsis and hypoperfusion, toxicity, obstruction, & parenchymal kidney disease. Background morbidities in the form of elderly, CKD, cardiac failure, liver failure, diabetes mellitus, vascular disease, nephrotoxic medication also contribute to insult. [4]

To prevent AKI: 4M 1. MONITOR PATIENTS

2. MAINTAIN CIRCULATION

3. MAINTAIN KIDNEY FUNCTION

4. MANAGE THE ACUTE ILLNESS.

AIMS AND OBJECTIVES

Etiology and clinical presentation of acute kidney injury varies based on the endemicity of the area. Aim of the study is:

- To prioritize the common etiologies of acute kidney injury in our hospital.
- To assess the outcome of acute kidney injury in respect to its varied etiologies.
- To predict the mortality rate of acute kidney injury in our hospital during the study period.

HISTORICAL REVIEW

AKI is an ailment that has afflicted humans from time immemorial.

WILLIAM BOWMAN (1816-1892) described bowman capsule and connection between glomerular capillary tuft and proximal tubules.

In 1888 DALAFIELD describes microscopic pathology of AKI. In 1917 during World War I AKI due to trauma reported as War Nephritis.

In 1941 LANDMARK ARTICLE by waters and brall in World War II in crush victims followed by impaired renal function and demonstrate widespread tubular damage and pigmented cast inside tubular lumen.

In 1951 HORMER W SMITH introduced the term acute renal failure in his textbook ``THE KIDNEY STRUCTURE AND FUNCTION IN HEALTH SCIENCE [5]

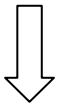
In 1967 Silverstein Lee Henderson introduced hemodialysis. In 1979 kraner describes continous AV Hemodialysis.

PHu in 2002 described sepsis induced AKI & advantage of hemofiltration over automated peritoneal dialysis.

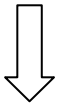
The term acute renal failure has replaced by acute kidney injury in 2004 as defined by RIFLE criteria .it also incorporates the entire spectrum of syndrome from minor change in kidney function to requirement of renal replacement therapy.

HISTORICAL MILESTONES :-

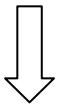
GALEN (EMPTY BLADDER) - (GREEK, ROMAN)



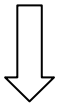
MORGAGNI, 1760 (ISCHURIA RENALIS) - (18^{TH} CENTURY)



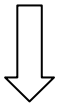
BRIGHT, 1888 (ACUTE BRIGHT'S DISEASE) - (19^{TH} CENTURY)



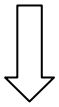
DAVIES, 1917 (WAR NEPHRITIS) - (20^{TH} CENTURY, BEFORE WORLD WAR)



BYWATERS AND BEALL, 1941 (CRUSH SYNDROME)- (20^{TH} CENTURY, AFTER WORLD WAR II)



SMITH, 1951 (ARF)



ADQI, 2004 (AKI)

DEFINITION AND STAGING OF AKI:

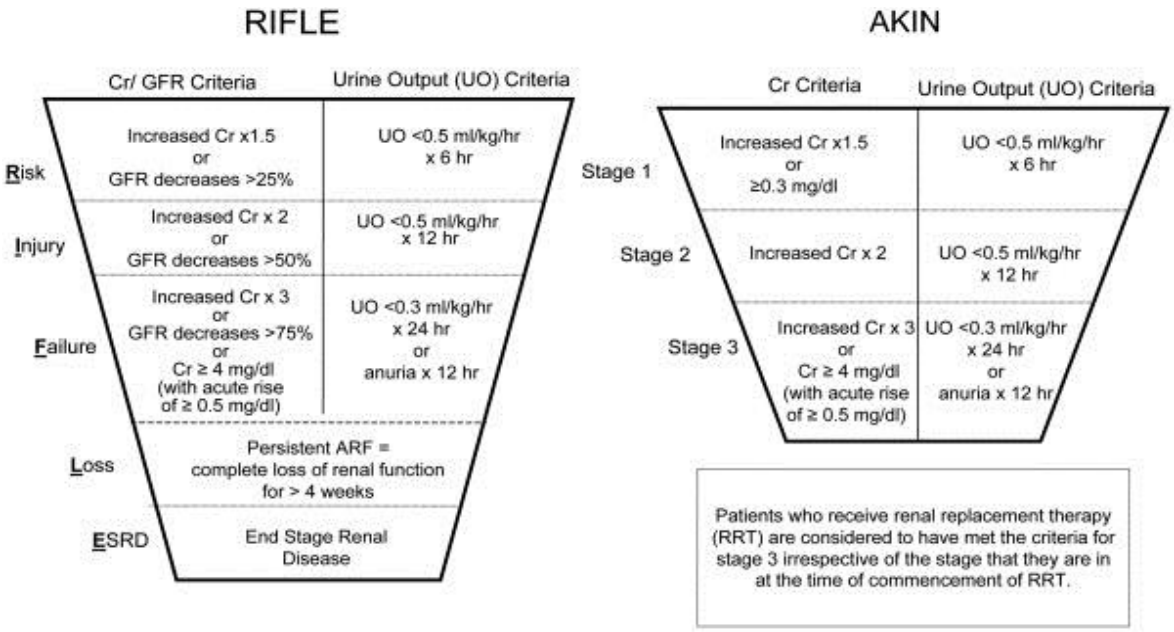
It is defined by any one of the following (KDIGO) [1]

Increase in serum creatinine by ≥0.3mg/dl (≥26.5µmol/l) within 48 hours.

Increase in serum creatinine to ≥1.5 time's baseline which is known or presumed to have occurred within the previous seven days.

Urine volume <0.5ml/kg/hr for more than six hours.

(RIFLE AND AKIN CRITERIA) :



EPIDEMIOLOGY:

The epidemiology of AKI is poorly characterized .The incidence depends upon précised population and definition of AKI. In 2001 AKI was diagnosed in 1.9% of hospitalized patients with RRT required in 7.5%of cases. Hospital mortality was 21.3% in patients who had AKI compared with 2.5% in patients not identified as AKI.

In a similar analysis during 1992-2001 with progressive increase incidence of AKI 11% per year over the 10 years studied -14.6 cases/1000 discharges in 1992 to 36.4 cases/1000 discharges in 2001 Rate of AKI increased from 0.4% in 1998 to 2.1% in 2002 ; but the mortality decreased from 40.4% to 20.35.

In multicenter trials 29,269 critically ill patients in 54 hospitals in 23 countries the period prevalence of AKI was 5.7% with 72.5% [(7)] of these patients required RRT.ICU mortality rate has 52% with an additional 8% mortality in patients after ICU discharge. The overall mortality of 60.3%.Among survivors 13.8% continues to require RRT at the time of hospital discharge.

AKI is synonymous with the older term acute renal failure (ARF) and replacing the ARF in recent literature. The term AKI was introduced in the year 2004 by Acute Dialysis Quality Initiative (ADQI)

TYPES OF ACUTE KIDNEY INJURY

Acute kidney injury is divided extensively in to three categories; they differ in pathophysiology and treatment.

1. PRERENAL AZOTEMIA : represents functional response to renal Hypoperfusion [7] and it is not associated with structural renal injury.

2. RENAL AZOTEMIA: diseases that directly involves renal parenchyma (Intrinsic AKI).

3. POST RENAL AZOTEMIA: diseases associated with urinary tract obstruction. (Post renal AKI).

CAUSES OF PRERENAL AKI:

HYPOVOLEMIA:

INCREASED EXTRACELLULAR FLUID LOSSES:

Hemorrhage Gastrointestinal fluid loss: vomiting, diarrhea, Renal fluid loss: diuretics, osmotic diuresis, hypoadrenalism, nephrogenic diabetes insipidus.

EXTRAVASCULAR SEQUESTRATION:

Burns, pancreatitis, severe hypoalbuminemia (hypoproteinemia)

DECREASED INTAKE: hypovolemia, altered mental status

LOW CARDIAC OUTPUT STATE:

Diseases of the myocardium or valves

Pericardial diseases (including tamponade)

Pulmonary hypertension

Massive pulmonary embolism

Impaired venous return (e.g.,abdominal compartment syndrome or positive pressure ventilation).

SYSTEMIC VASODILATION:

Sepsis

Antihypertensive

Anaphylaxis.

RENAL VASOCONSTRICTION:

Hypocalcaemia

Catecholamine

Calcineurin inhibitors

Amphotericin B.

IMPAIRMENT OF RENAL AUTOREGULATORY RESPONSES:

Cyclooxygenase Inhibitors, Angiotensin-Converting Enzyme Inhibitors Or Arbs, Hepatorenal Syndrome.

PATHOPHYSIOLOGY:

In prerenal AKI, restoration of normal renal perfusion results in early recovery of Renal function .However sustained renal hypoperfusion [7] results in irreversible renal injury. Decreased renal perfusion or effective arterial blood volume depletion is described by activation of sympathetic nervous system and renin angiotensin System.

Increased angiotensin II level constricts the postglomerular and preglomerular arteriole and it is opposed by vasodilator prostaglandins, [9] the predominance of postglomerular vasoconstriction maintain intraglomerular capillary pressure to normal and maintains near normal GFR.

Hemodynamic factors, increased levels of angiotensin II, activation of Sympathetic system will increase proximal tubular sodium and water reabsorbion.

Aldosterone and vasopressin secretion [10] will leads to increased sodium, urea and water reabsorbing in distal convoluted tubules .Thus physiological response

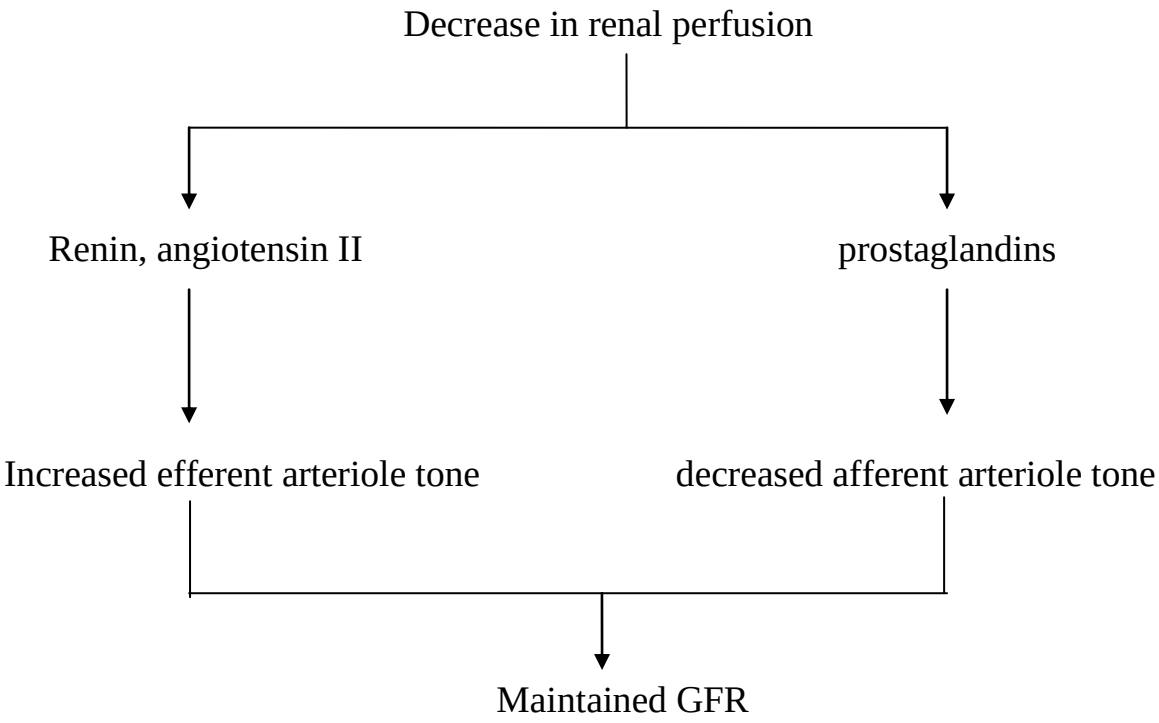
leads to concentrated urine with decreased sodium concentration.

In patients with prerenal acute kidney injury the regulatory mechanism are unable to compensate fully for more severe degree of hypoperfusion it results in decline in glomerular filtration rate.

Autoregulation [11] is the first line defence of kidney against fluctuations of arterial blood pressure. When the renal perfusion decreases, the afferent arteriole senses the degree of stretch and thus relaxes. This is called myogenic reflex.

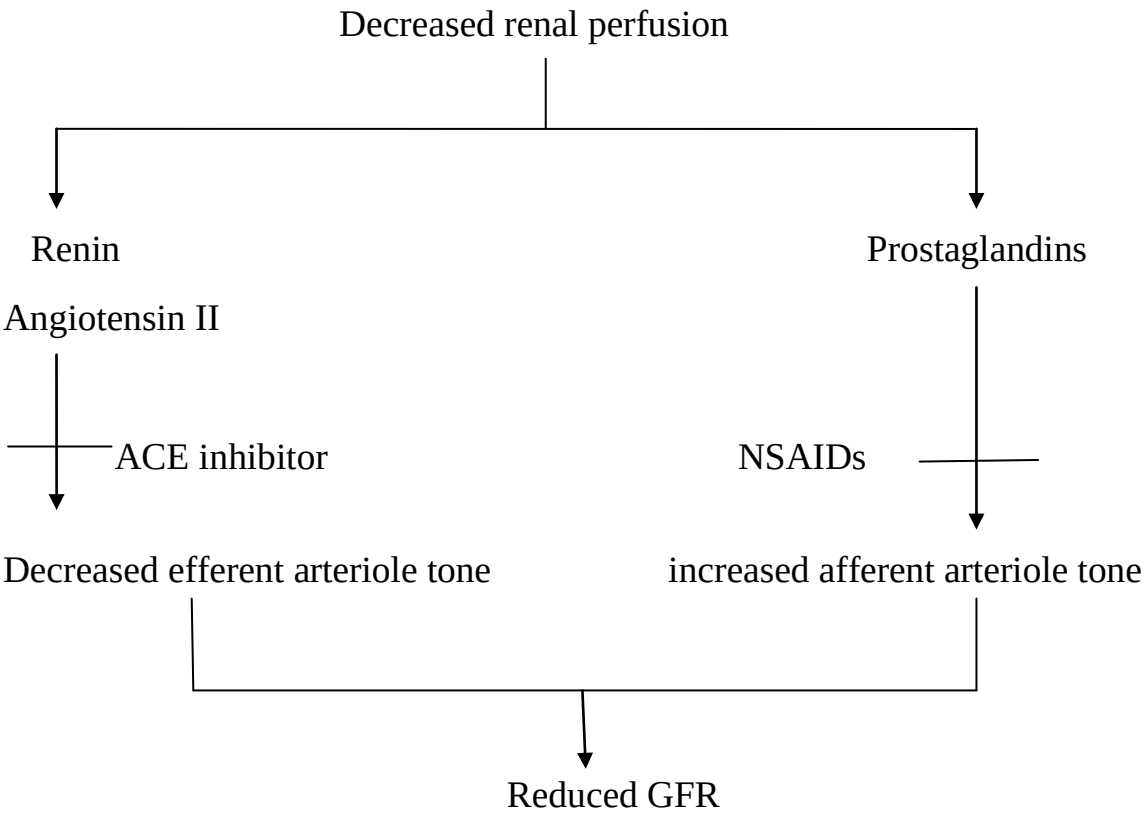
TUBULO-GLOMERULAR FEEDBACK also plays an important role in auto-regulation. Macula densa presenting in the cortical collecting ducts senses the decrease in solute delivery to the distal tubules and leads to afferent arteriole dilatation by releasing nitric oxide.

AUTOREGULATION



Autoregulation and other intrinsic compensatory mechanisms fails once the systolic blood pressure falls below 80 mmhg **.**

FAILURE OF AUTOREGULATION



The classic feautures are

Low urine sodium concentration <20mmol/lit

FeNa < 1%

Low fractional excretion of urea <30%

Increased urine osmolality.

CAUSES OF INTRINSIC ACUTE KIDNEY INJURY:

RENOVASCULAR OBSTRUCTION : (bilateral or unilateral in the setting of one kidney)

Atherosclerotic plaque.
Dissecting aneurysm
Large vessel or small vasculitis.
Thrombosis.
Embolism.
Obstruction of renal vein- thrombosis and compression.

DISEASES OF THE GLOMERULAR VASCULATURE:

Glomerulonephritis /vasculitis

Disseminated intravascular coagulation

Preeclampsia

Thrombotic microangiopathy.

Malignant hypertension.

Collagen vascular diseases- systemic lupus erythematosus, scleroderma

ACUTE TUBULAR NECROSIS

Ischemia- [12]

causes are the same as for prerenal ARF, but generally the effect is more severe and more prolonged.

Toxins -EXOGENOUS - calcineurin inhibitors.

Antibiotics –Aminoglycosides [18,19]

Radiocontrast [16]

Antifungals -Amphotericin B[19]

Ethylene glycol

Chemotherapy – cisplatin [20]

ENDOGENOUS- rhabdomyolysis

hemolysis

ACUTE INTERSTITIAL NEPHRITIS:

Allergic: Antibiotics:βlactams,Sulfonamides,quinolones,rifampin.

NSAID, Diuretics.

Infection: Pyelonephritis -bilateral

Infiltration: Lymphoma,Leukemia,Sarcoidosis.

Inflammatory: Sjögren's syndrome.

Tubulointerstitial nephritis with uveitis.

Intratubular obstruction: Endogenous- Myeloma proteins, [21]

Uric acid (tumor lysis syndrome),[22]

Systemic oxalosis.

Exogenous-Acyclovir, ganciclovir, methotrexate, indinavir.

PATHOPHYSIOLOGY:

Intrinsic AKI classified into Acute glomerular, interstitial and tubular injury. Correction of underlying cause does not result in complete recovery

ACUTE TUBULAR NECROSIS:

Acute tubular necrosis is the most common cause by the precipitating events such as ischemic and nephrotoxic process but frequently ATN is multifactorial such as acute illness with sepsis, hyponatremia, nephrotoxic drugs. [15] Clinically ATN had intial oliguric phase within 24 hours of insult and last for 1-3 weeks followed by diuretic phase indicating renal recovery. Mortality rate is due to comorbid illness.

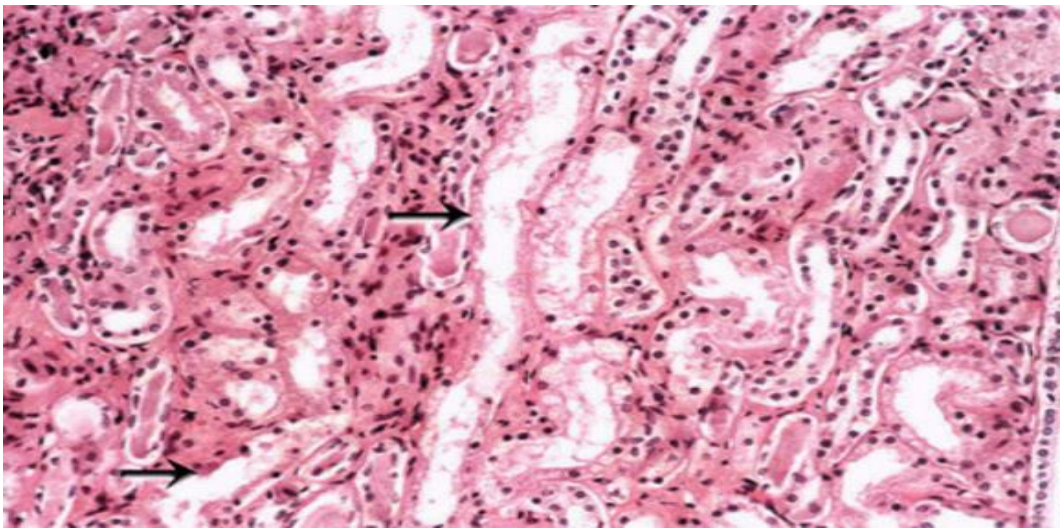
The surviving patients recover kidney function but complete recovery may not Occur.

The urine sediment shows muddy brown granular cast.

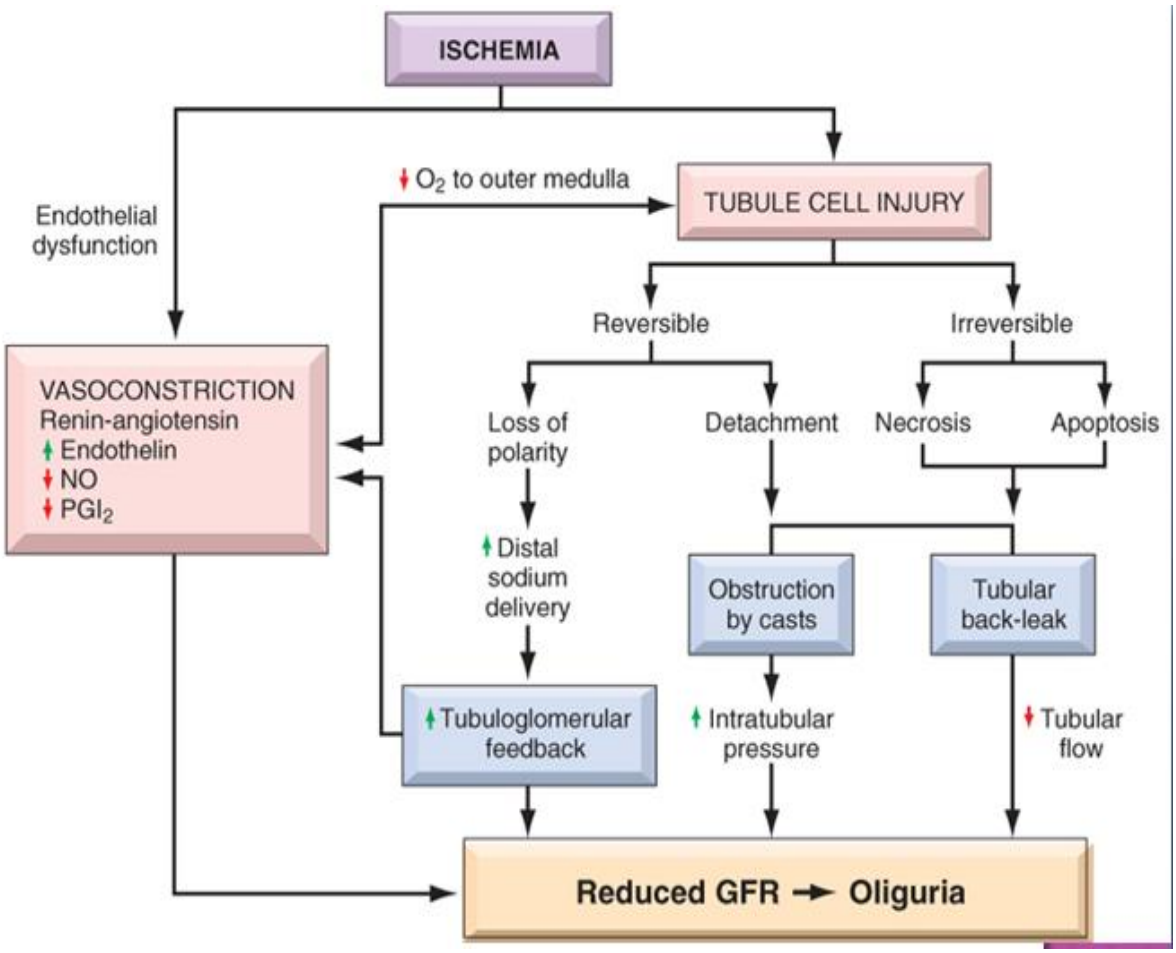
Urinary sodium >40mmol/lit

FeNa in excess of >30%

Sloughing of cells, which is responsible for the formation of granular casts, is a feature of acute tubular necrosis.



Photomicrograph of a renal biopsy specimen shows renal medulla, which is composed mainly of renal tubules. Patchy or diffuse denudation of the renal tubular cells with loss of brush border is observed, suggesting acute tubular necrosis as the cause of acute renal failure.



ISHAEMIC ATN; [12]

Vasoconstriction.

Desquamation of tubular cells.

Intraluminal tubular obstruction ends in tubular backleak.

Local production of inflammatory mediators ends in interstial inflammation

small vessel obstruction &local ischemia.

NEPHROTOXIC ATN: [17]

Mechanism	Agent
Direct tubular cell injury	Aminoglycosides, vancomycin, cisplatin, radiocontrast agents, amphotericin B, heavy metals, foscarnet, cyclosporin
Endothelial cell injury	Cyclosporin, cocaine, tacrolimus, mitomycin C, quinine
Vasoconstriction	NSAIDs, radiocontrast agents, cyclosporin, amphotericin, heme pigments
Efferent arteriolar vasodilation	Angiotensin-converting enzyme inhibitors
Crystalluria	Ethylene glycol, sulfonamides, uric acid (tumor lysis syndrome), triamterene, acyclovir, methotrexate, protease inhibitors
Interstitial nephritis	Any drug
Glomerulopathy	Gold, penicillamine, NSAIDs
Hemolytic uremic syndrome	Conjugated estrogens, cyclosporin, tacrolimus, mitomycin, cocaine, quinine

SEPTIC ACUTE TUBULAR NECROSIS: [13,14]

Systemic and renal hypoperfusion

Endotoxins

Activation of inflammatory mediators

Microvascular endothelial damage.

ACUTE INTERSTITIAL NEPHRITIS:

Lymphocytic infiltration of interstitium.Clinically it presents as fever, rash, eosinophilia, eosinophiluria in only 10 to 30%.

Most common cause are NSAIDS, antibiotics, infection, malignancy and systemic disease.

Urine findings: sterile pyuria, WBC casts, haematuria, Non nephrotic range proteinuria

Diagnosis: renal biopsy.

ACUTE GLOMERULONEPHRITIS:

Acute glomerulonephritis and RPGN- Most common causes are post streptococcal glomerulonephritis, endocarditis, other infections.

The pathology is related to damage to glomerular basement membrane and glomerular bleeding.

Urine shows dysmorphic RBC, RBC casts are pathognomonic.

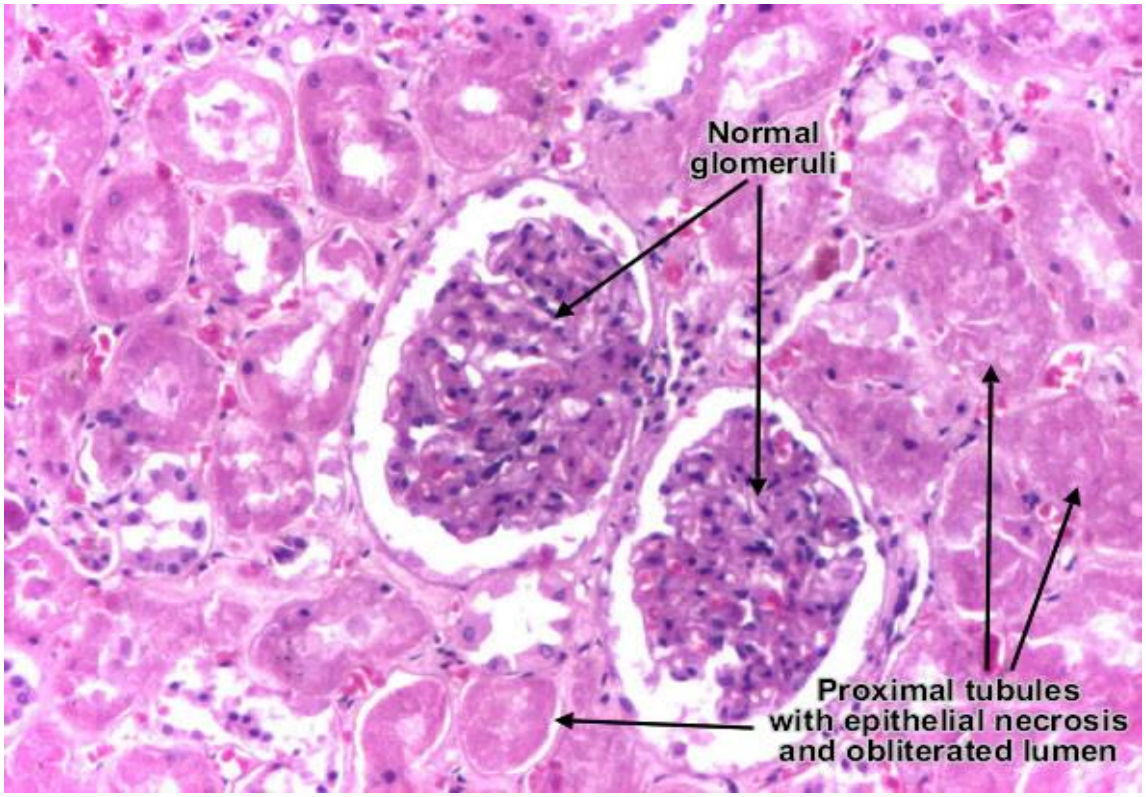
Serology: complement assays, markers of hepatitis B, hepatitis C,

Anti Streptococcal antibody, ANA, ANCA, anti GBM antibody.

Defnite: renal biopsy which shows proliferative changes in glomerulus and crescentic formation.

RADICONTRAST NEPHROPATHY: [16]

It is a rapid decline in renal function after radiocontrast administration. The serum creatinine begins to rise 24 to 76 hours after contrast administration and peaks at 3 to 5 days and return to baseline within 3 to 5 days.



Histopathology of AKI associated with toxins and venoms.

POSTRENAL AKI :[22]

Post renal AKI results from obstruction of urinary collecting system

CAUSES:

UPPER TRACT OBSTRUCTION:

Intrinsic: Nephrolithiasis

Papillary necrosis

Blood clots

Transitional cell carcinoma

Extrinsic: Retroperitoneal or pelvic malignancy

Retroperitoneal adenopathy

Retroperitoneal fibrosis

Endometritis

Abdominal aortic aneurysm

Surgical injury

LOWER TRACT OBSTRUCTION:

Bladder: Neurogenic bladder

Transitional cell carcinoma

Blood clot

Bladder calculus

Prostate: prostate cancer

Benign prostate hypertrophy

Urethra: Strictures, Phimosis, Urethral valves

The pathophysiology of AKI leads to increased hydrostatic pressure in the renal Tubules leads to decreased glomerular filtration rate .Inspite of effective advances in renal replacement therapy and Supportive measures, mortality rates in AKI exceed 50%.

The poor prognostic factors of acute kidney injury are Age >65 years, Acute Respiratory failure, cardiac failure, Multi organ dysfunction syndrome. [23,24]

Groeneveld et al studied that 490 patients admitted to intensive medical care unit With AKI had mortality >60%.**Gurucharan et al** found that incidence of AKI is Increased in burns and surgical ICU patients. [24]

Nuraula et al found that 75 % Patients had medical cause for acute kidney injury, the most common being Septicemia, malaria, drugs, volume depletion, post transplant renal failure.

SNAKE BITE INDUCED AKI:

ACUTE KIDNEY INJURY complicates 5 to 30% of snake bite majority of snake bite induced acute kidney injury are due to viper bite.

WHO estimates that 1,25000 Death every year worldwide. In India it accounts for 10,000 deaths every year.

Snake bite induced AKI has diagnosed according to following criteria.

Glomerular filtration rate rate of < 60 ml/m2 within 72 hours after snake bite. Glomerular filtration rate was estimated with Cockcroft gault equation.

Snake bite induced AKI was increased in the following group of patients.

Deranged haemostatic profile:

An abnormal whole blood clotting time

Prothrombin time/activated partial thromboplastin time above 1.5 times normal.

BLOOD PARAMETERS:

Created red blood cells in peripheral smear

Increase in serum creatinine of> 30% of baseline

Proteinuria

Raised D dimer

Low platelets < 1 lakh/m3.

HYPOTENSION AND ALTERD HAEMODYNAMICS:

Haemarrhage

Vasodilation

Increased vascular permeability

Direct or indirect cardiotoxicity

PIGMENT NEPHROPATHY:

Haemoglobinuria after haemolysis

Myoglobinuria after rhabdomyolysis.

PATHOGENESIS OF ACUTE KIDNEY INJURY:

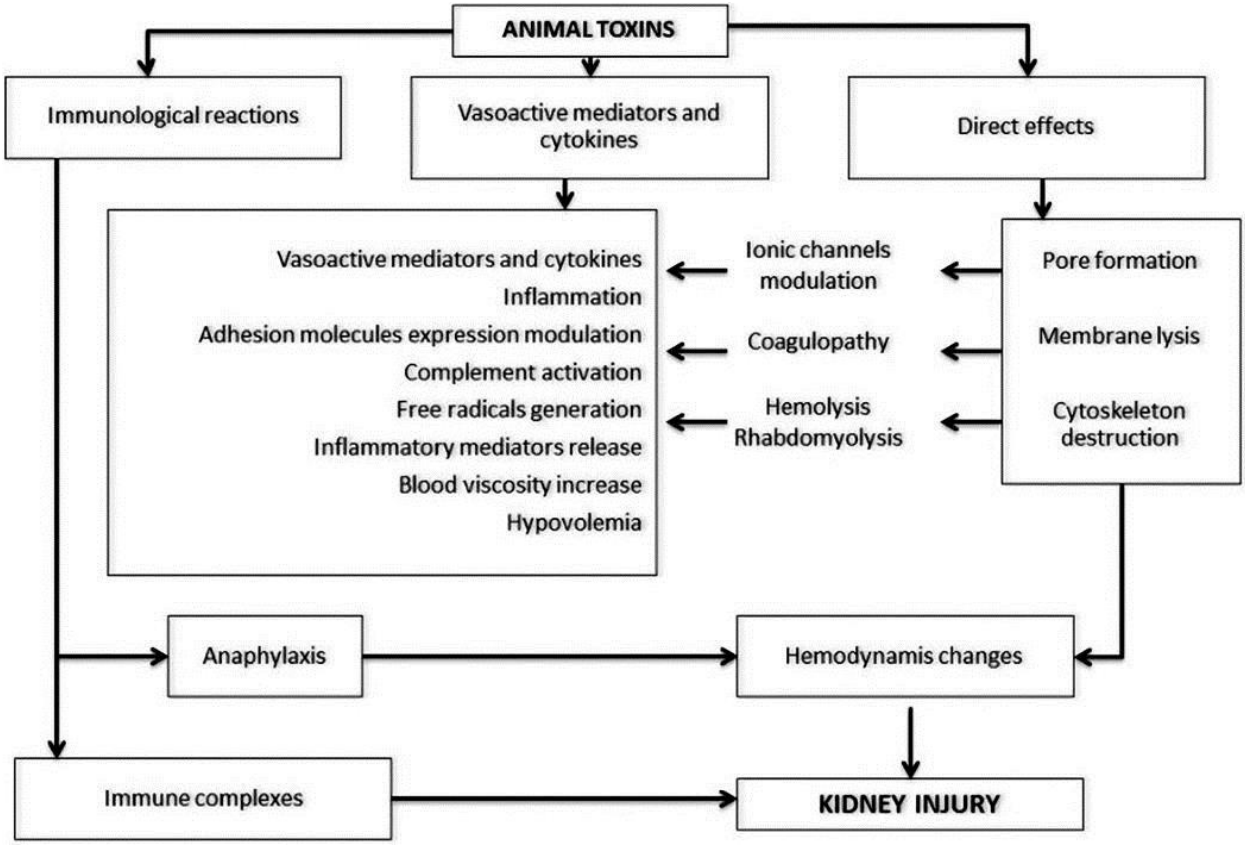
More than 100 different kinds of peptides or proteins,lipids isolated from venom.

Activation or inhibition of various coagulation proteins, endothelial disruption by phospholipase A2, serine protease, metalloproteinase and lectins leads to cogulopathy.

Haemarrhage leads to spontaneous bleed by directly injuring the vascular endothelium. digestive hydrolases,hylaurinadase, and polypeptide

cytotoxin leads to local tissue necrosis. Permeability factors leads to increased extravasation of Plasma from intravascular smooth muscle leads to hypotension.

GUPTA et all., found that in 121 patients with snake bite 15 patients developed Acute kidney injury out of these 7 patients on conservative treatment and 8 needs dialysis.



ACUTE GASTROENTERITIS INDUCED ACUTE KIDNEY INJURY:

AKI due to AGE is common in adult and elderly people. Kidneys receive one fifth of cardiac output .Due to severe diarrhea loss of fluid and electrolytes leads to Renal hypoperfusion and also intense vasoconstriction during shock. tubular dysfunction also occurs due to shedding of epithelial cells. Most commonly Presents with oliguria, metabolic acidosis due to bicarbonate loss, hypokalemia.

MC histological lesion acute tubular necrosis. acute cortical necrosis also occurs.

Mortality due to AKI is mainly due late referral, severe acidosis, anuria, metabolic Encephalopathy and mutiorgan failure.

Chugh et all reported 63% of medical renal disease most commonly due to AGE the use of better sanitation, early treatment, decrease the incidence of post diarrheal acute kidney injury from 23% to less than 10% in the last 20 years.

ACUTE KIDNEY INJURY IN SEPSIS:

Sepsis accounts for more than 40% of critically ill patients. In sepsis the Development of AKI is a higher predictor of mortality. Increase risk of AKI

Mortality in patients with age >65 years, hypertension, higher APACHE Score, severe anemia, hyperphosphaetemia, hyperkalemia .

In pregnancy common cause of AKI is septic abortion in early Pregnancy, puerperal sepsis in postpartum period. The pathogenesis is most

Commonly due to ischemia, reperfusion injury, direct inflammatory injury, coagulation and endothelial cell dysfunction and apoptosis.

DRUG INDUCED AKI

HIGH RISK COMBINATION FOR ACUTE KIDNEY INJURY:

Volume depleted state: aminoglycosides

Amphotericin B [19]

Diuretics

Haeme pigments

Preexisting renal disease or bilateral renal arterial disease: ACE inhibitors, Angiotensin receptor blockers , Hypertension, diabetes, elderly, congestive heart failure, jaundice: NSAID,Radiocontrast agents.

FEVER INDUCED AKI: [27]

AKI is common in malaria. In endemic areas incidence of AKI >4 % mortality rates >45% in falciparum malaria.[29]

The pathogenesis is impaired microcirculation due to parasitized red blood cells, Hypovolemia, disseminated intravascular coagulation, intravascular haemorrhage , jaundice.

In a study panda sk et al., found that AKI had been associated with 20 % of malaria. Renal failure was seen within 5 to 7 days from onset of fever. Other causes are leptospirosis, dengue, typhoid, and viral haemorrhagic fever.

Most common cause of AKI in india :

Diarrheal disease

Sepsis

Drug induced

Hospital acquired

Fever- malaria, leptospirosis, viral hemorrhagic fever.

PATHOGENESIS OF ACUTE KIDNEY INJURY:

The pathogenesis of AKI is complex involving renal injury and repair.

Kidneys are susceptible to ischemia and toxins leads to vasoconstriction, endothelial damage and activation of inflammatory process. Inflammation results.

In reduction in local blood flow to the outer medulla with alteration in tubule function and viability.

With ischemia & reperfusion, endothelial cells express a number of adhesions Molecules which are counter receptors on leucocytes. Numerous vasoactive. Mediators that are released with injury such as nitric oxide may also affect leucocytes endothelial interactions.

There is obstruction of the microvascular as well as enhanced vasoconstriction tubules generate proinflammatory and chemotactic cytokines.

Injection of stromal cells is protective against renal injury which is assessed by serum creatinine measured 24 hours after ischemia. It is thought it is due to intra renal paracrine effects to decrease inflammation or by systemic immune modulation. The epithelial cells which replace the lost cells derive from surviving tubular epithelial cells.

CLINICAL COURSE OF ACUTE KIDNEY INJURY: [1,2]

The course of AKI is characterized by three phases

INITIATION PHASE (HOURS TO DAYS)

Initial period of renal hypoperfusion during which ischemic injury is evolving.

GFR decline because of decrease in glomerular ultrafiltration rate, presence of glomerular filtrate within tubules. and backleak of glomerular filtrate through injured tubular epithelium. This phase lasts for hours to days.

Restoration of renal perfusion or elimination of nephrotoxin may recover or limit the kidney injury.

MAINTANENCE PHASE:

The phase lasts for one to two weeks during which renal injury is established . GFR stabilizes at nadir (5 to 10 ml/min).urine output will be very low.

Uremic complications will appear in this phase.

RECOVERY PHASE:

Characterized by repair and regeneration of renal parenchymal cell particularly tubule epithelial cell and gradual return of glomerular filtration rate. The recovery phase may be complicated by diuretic phase owing to marked excretion of retained salt and water.

Sodium handling and concentration functions are the last function to recover particularly in elderly. In general recovery of renal function is less complete in older patients than young.

PROGNOSIS: [3]

The prognosis of acute kidney injury depends upon causes and severity of disease. Overall prognosis in acute kidney injury is poor. Morbidity depends upon severity of injury and underlying disease .Patients requiring dialysis is at higher risk.

Factors influencing patient survival in acute kidney injury:

Etiology of acute kidney injury

Severity of AKI as evidenced by oliguria, maximal serum creatinine and need for RRT

Number and severity of coexistent illness.

Age

Complications

Infection-sepsis, pulmonary infection, hypercatabolism.

Non infectious causes - heart failure, respiratory failure, gastrointestinal disease, advanced liver disease, pancreatitis and burns.

Several studies have identified that age has an adverse prognostic factors in acute kidney injury. Age more than 60 years has poor prognostic indices than younger patients. However studies have shown

that sex has no predicting value. Among the medical cause AKI induced by aminoglycosides, nephrotoxic agents have good prognosis and acute kidney injury following sepsis has poor prognosis.AKI due to medical causes has better prognosis than surgical causes.

kaufmon et al found that despite a rapid reversal of AKI in prerenal and postrenal patients ,mortality is still high probably because of coexisting diseases like shock , acute and chronic cardiac disease, respiratory failure, mechanical ventilation , pneumonia, chronic lung disease .Vascular diseases have poor outcome in acute kidney injury. Overall oliguric AKI has a worse prognosis.

CLINICAL FEAUTURES OF ACUTE KIDNEY INJURY:[1]

The common symptoms are:

Oliguria- urine output less than 400ml/day

Anuria

Convulsion

Breathlessness

Obstructive urinary symptoms

Nausea and vomiting

Weakness

Physical signs:

PRE RENAL AKI:

Volume depletion- absolute or postural hypotension

Low jugular venous pressure

Dry mucous membrane

Decreased effective circulatory volume- heart failure

Hepatic failure

RENAL AKI:

Renal artery thrombosis- flank or abdominal pain

Atheroembolic disease- retinal plaques, palpable purpura, livedio reticularis.

Renal vein thrombosis- history of nephritic syndrome or pulmonary embolism.

Disease of small vessel & glomeruli- new cardiac murmur (postinfectious) skin rashes/ ulcers(lupus) Sinusitis (anti GBM disease) Lung haemorrhage (anti GBM , ANCA, lupus)

Haemolytic uraemic syndrome - fever, neurologic abnormalities.

Malignant hypertension - evidence of end organ damage such as headache, blurring of vision .heart failure, left ventricular hypertrophy.

Ischemia- recent hemorrhage or hypotension.

Exogenous toxins - recent exposure to nephrotoxic antibiotics or chemotherapy in coalition with sepsis or volume depletion. Exposure to radiocontrast in association with volume depletion, chronic kidney disease, diabetes.

Endogenous toxins- rhabdomyolysis (trauma, immobilization, post ictal state) haemolysis (fever or evidence of blood transfusion)

Tumour lysis- chemotheraphy

Multiple myeloma- age more than 60 years, fatigue, malaise, lowbackache.

Ethylene glycol – alcohol abuse, altered mental status.

Disease of tubulointerstitium- fever, rash, arthralgia.

POSTRENAL AKI: [30,31]

Increased urinary urgency, frequency, dysuria &dribbling

Recurrent urinary tract infection or renal calculi

Acute onset of anuria and polyuria

Prostate ,intraabdominal, cervical , retroperitonel or pelvic tumours.

Previous radiation therapy to abdomen

History of vaginal bleeding, subtotal hysterectomy.

LABORRATORY INDICATORS : [32]

Haemoglobin, complete blood count, erythrocyte sedimentation rate.

Blood urea, serum creatinine, serum electrolytes.

Serum albumin, total protein.

Liver function tests. Peripheral smear.

Diagnostic indices:

Prerenal AKI - high BUN /serum cretinine >20:1 is suggestive.

Elevated LDH - renal artery thrombosis.

Eosinphilia, hypocomplement - atheroembolic disease.

Small vessel vasculitis – ANA , ANCA,anti GBM antibody,hepatitis serology, blood culture,complement.

Haemolytic uraemic syndrome - schistocytes on peripheral smear, increased LDH, anaemia, thrombocytopenia.

Rhabdomyolysis – increased myoglobin & creatine kinase.

Haemolysis – pink plasma, increased LDH.

Tumour lysis - hyperurecemia, hypocalcemia, increased LDH.

Multiple myeloma – circulating monoclonal spikes, anaemia.

Ethylene glycol ingestion – high anion metabolic acidosis.

URINARY INDICES IN ACUTE KIDNEY INJURY:

PRERENAL AKI:

hyaline casts

FENa < 1%

Urine sodium < 10 mmol/lit

Specific gravity > 1.018

Urine urea nitrogen / plasma urea nitrogen > 8.

Urine cretinine / plasma creatinine >40.

Urine osmolality >500.

Renal failure index < 1.

INTRINSIC AKI:

Acute tubular necrosis – muddy brown or tubular epithelial casts

FENa >3 %

Urine sodium > 40 mmol/lit

Specific gravity-1.010

Fractional excretion of urea - < 60 %

Acute interstitial nephritis – white blood cell cast

FENa > 1%

Haematuria

Eosinophilluria.

Acute glomerulonephritis –urine sodium < 20 mmol/lit

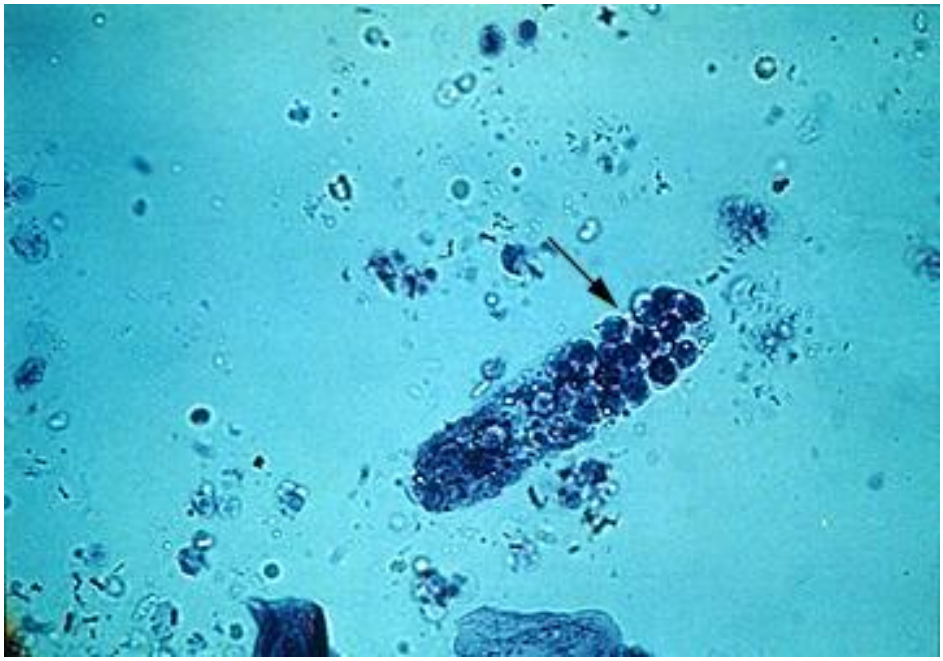
FENa < 1 %.

Dysmorphic red blood cells / RBC casts.

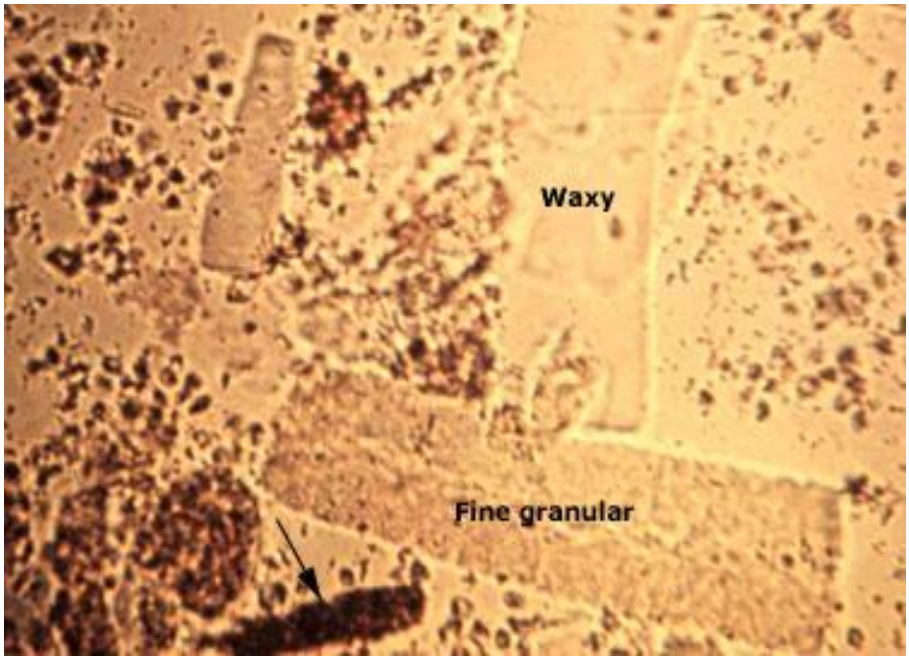
The following table shows some differentiating features between AKI and CKD

FEATURE	AKI	CKD
Antecedent history of renal disease	Absent	Present
Prior sustained elevation of Creatinine for > 3 months	Absent	Present
Anaemia	Usually absent at onset	Present
Elevated serum phosporous, PTH. Decreased serum calcium	Absent (in early phase)	Present
Neuropathy	Absent	Present
Band keratopathy	Absent	Present
Renal bone disease	Absent	Present
Small kidneys on USG	Absent	Presen
Tolerance to azotemia, acidosis	Absent	Present
Stability of azotemia	Absent(daily rise)	Present

WBC CAST:

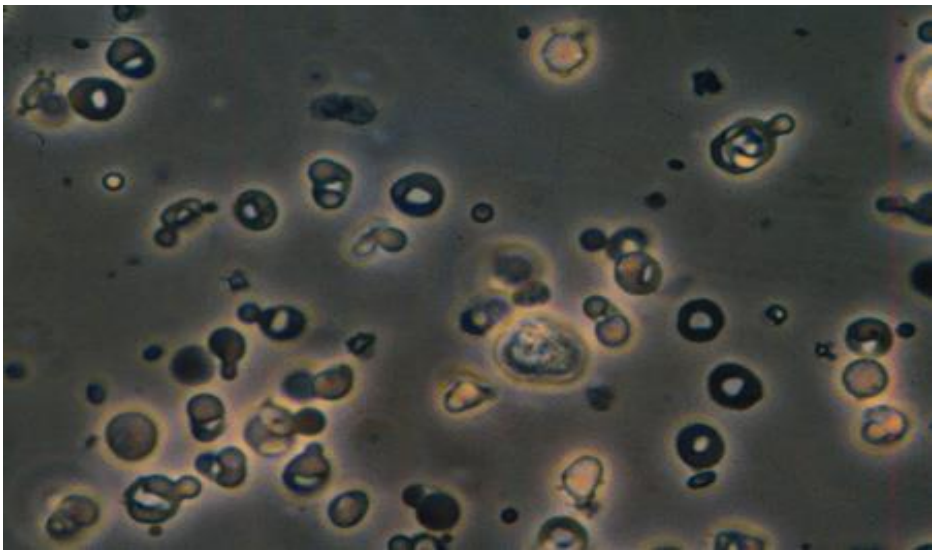


GRANULAR CAST:



RBC CAST:

DYSMORPHIC RBC



IMAGING PROCEDURES IN THE DIAGNOSIS OF ACUTE KIDNEY INJURY:

Ultrasonography :To assess the kidney size, calculi, hydronephrosis, renal or retroperitoneal mass.

Intravenous urography: for assessing kidney size,function , hydronephrosis, hydroureter , bladder size ,bladder outlet obstruction .

Radionucleotide scans : for assessing blood flow & function,gallium 67 scan for acute interstitial nephritis.

Renal biopsy :

Indications : acute kidney injury of unknown etiology

AKI associated with glomeulonephritis, nephrotic syndrome, or vasculitis where acute theraphy is contempted.

AKI with interstitial disease without a definite etiology.

Prolonged acute kidney injury for more than 28 days

AKI associated with systemic disease : SLE, wegners granulomatosis

MARKERS OF KIDNEY FUNCTION:-

At present there is no single marker valid that can predict the renal function. Serum creatinine and BUN are commonly used to estimate the renal function.

UREA AND UREA CLEARANCE

Urea elimination by kidney is a complex process. Hence BUN is a less useful marker of kidney function plasma level of urea is impacted by many factors other than GFR . Non renal causes for exalted BUN level are GIT bleeding, steroid use, total parenteral nutrition. Also malnutrition, chronic liver disease can cause decrease in BUN level due to reduced production.

CREATININE AS A MARKER FOR KIDNEY FUNCTION:-

Creatinine is a metabolic product of creatine and the major sources are skeletal muscles, dietary meat. Daily production of creatinine ranges from 20 to 25 mg / kg / day in males and 15 to 20 mg / kg /day in females.

Creatinine is removed by both glomerular filtration and proximal tubular secretion. In healthy individuals, >90% of creatinine removal is by glomerular filtration and the rest is by tubular secretion. But when renal function starts decreasing, the proportion of creatinine which is eliminated by tubular secretion increases up to 50%. (38)

Glomerular filtration rate (GFR):

GFR is defined as the sum of the filtration rate of all functional nephrons. The normal value is around 125 ml/min/1.73m^2 for men and 100 ml/min/1.73m^2 for women. GFR is measured by the following formula,

$$GFR = U * V / P.$$

Where, U is the concentration of the substance in urine

P is the concentration of the substance in plasma

V is urine flow rate.

The substrate used should be biologically inert, freely and completely filtered by the glomeruli, neither secreted nor absorbed by tubules, and not degraded by the kidneys.

Inulin was once measured gold standard of exogenously administered markers of GFR. But, a number of factors like scarcity, high cost, problems related to urine collection to determine inulin clearance limits the usefulness of inulin as a marker of GFR. Creatinine is routinely used to calculate GFR.

Estimation of creatinine clearance & GFR by creatinine based equations:

Cock croft- gault formula, modification of diet in renal disease study (MDRD) equation are the two widely used creatinine based equations for the estimation of GFR in adult.

- COCKCROFT – GAULT EQUATION[39]:

$$\text{Est. creatinine clearance} = \frac{(140 - \text{age}) \times \text{body weight} \times 0.85 \text{ (if female)}}{72 \times \text{plasma creatinine}}$$

- MDRD EQUATION :

Est. GFR = $170 \times (PCr)^{-0.999} \times (age)^{-0.175} \times (0.762$ if female) $\times (1.180$ if African American) $\times (BUN)^{-0.170} \times (albumin)^{+0.318}$.

Cockcroft- gault and MDRD equations are important as they can underestimate GFR with normal renal function and overestimate GFR with severe renal dysfunction.

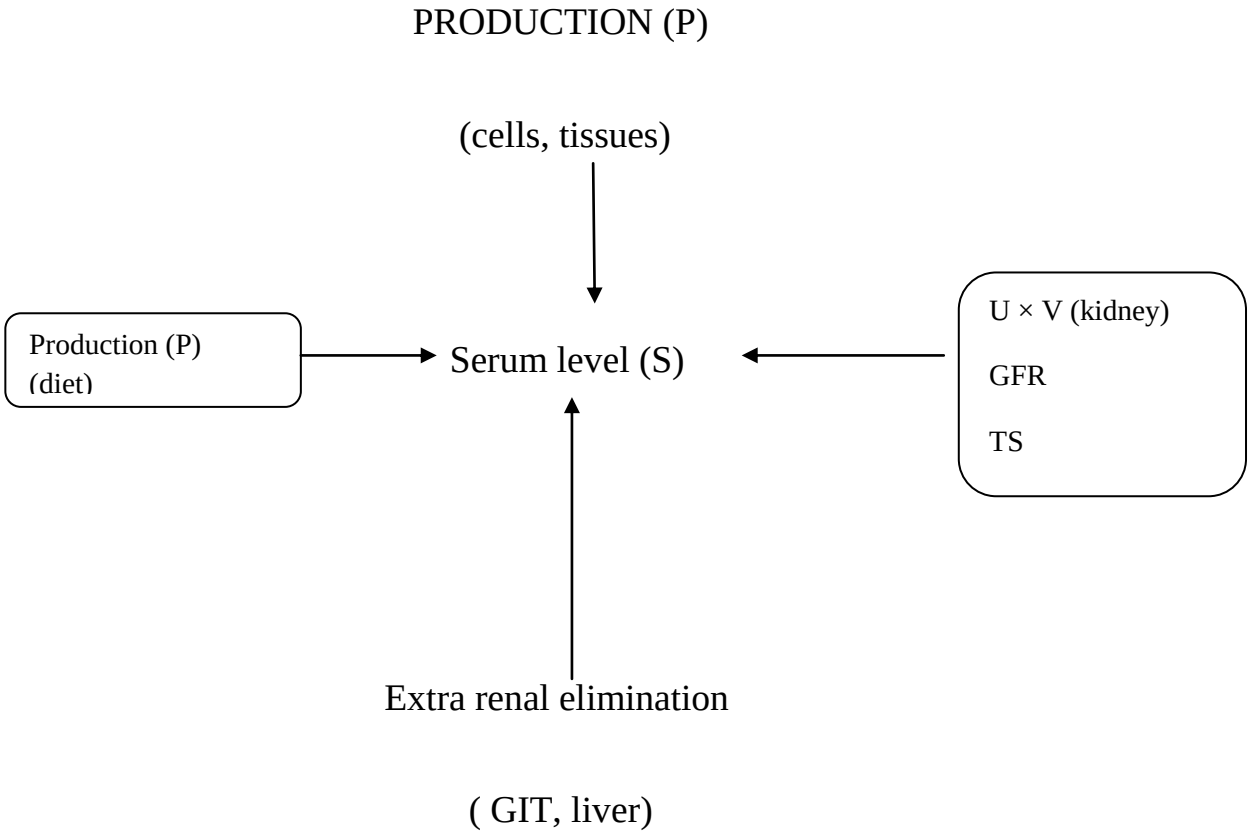
LIMITATIONS OF CREATINIE AS A MARKER FOR GFR:

Creatinine based GFR equations tend to over estimate renal function. so, when using creatinine to assess renal function the following limitations should be considered.

1) Creatinine production relay upon certain factors of a patient like body mass, race, age etc…
2) The change in plasma creatinine does not correlate with decline in renal function in a linear fashion.
3) The changes in creatinine in an individual should be interpreted based on the baseline creatinine value. For this a base line normal value is essential which is not available in most cases.
4) Creatinine level can also be altered by substances that interfere with the tubular secretion of creatinine. For example, drugs like trimethoprim, cimetidine blocks tubular secretion and hence can increase creatinine level.
5) Creatinine is a useful marker of GFR in the steady state only. In acute kidney injury it does not correlate with the sudden changes in GFR.

So, changes in serum creatinine level lag several days behind actual changes in GFR. Also the alterations in the level of serum creatinine for small changes in GFR are not so sensitive or specific. Hence many other low molecular weight serum proteins have been investigated as suitable endogenous markers of GFR

DETERMINANTS OF SERUM LEVEL OF CREATININE:



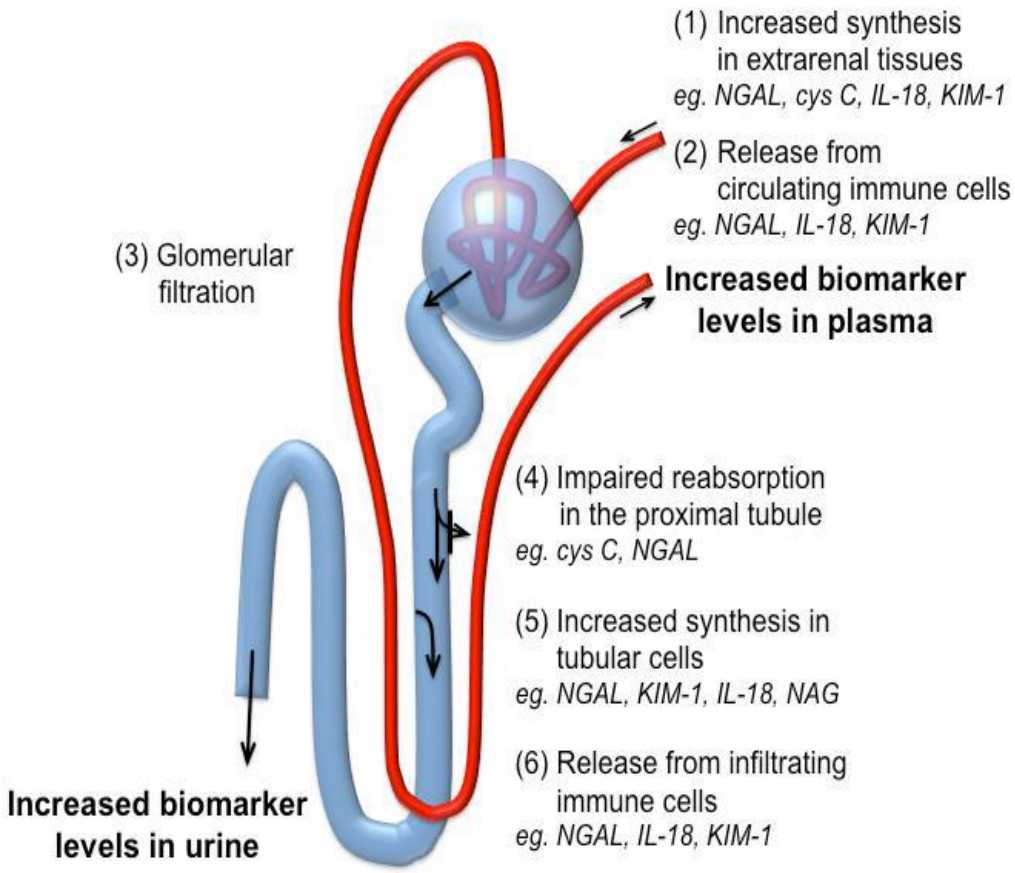
Where U × V denotes renal excretion.

(ie) the serum levels are determined by the interaction between production, excretion of creatinine.

BIOLOGICAL MARKERS OF ACUTE KIDNEY INJURY:

An abrupt change in **serum creatinine** , the most common indicator of AKI is strongly associated with poor outcome across multiple clinical trials.AKI presents as array of derangements by underlying precipitant, variable anatomical site of involvement, metabolic complication of unregulated inflammation , oxidative stress and insulin resistance.

The newly proposed markers such as urine IL 18,NGAL, L–FABP, KIM-1 , π-GST ,NAG and cystatin C differentiate between established AKI and control patients without acute elevation serum creatinine.



URINE CYSTATIN C:

Reabsorbed through megalin receptor in proximal tubules

Poor AKI predictor

Sepsis per se increase urine cystatin levels due to competitive inhibition of megalin receptor due to increased urine albumin in sepsis,

KIDNEY INJURY MOLECULE (KIM -1) [49]

Transmembrane glycoprotein expressed on epithelial cells after injury

Involved in the cellular repair process

Moderate ability to predict acute kidney injury

INTERLEUKIN 18: [51]

It is a pro-inflammatory cytokine secreted by the proximal tubular cells and leucocytes

Varying ability to predict AKI .

N-ACETYL β-d – GLUCOSAMINIDASE (NAG):

It is a lysosomal enzyme found in several human cells including tubular epithelial cells > 130 dalton not fitered , elevated urine levels in several acute renal diseases

Moderate ability to predict AKI.

URINARY LIVER TYPE FATTY ACID BINDING PROTEIN (L-FABP)

It is a fatty acid binding protein , molecular weight of approximately 14 Kda, distributed confined to the proximal tubular epithelial cells.

In healthy human kidney, LFABP is reportedly found in the cytoplasm of proximal tubular epithelial cells and is rapidly released in to the tubular lumen in response to ischemia and oxidative stress.

NEUTROPHIL GELATINASE ASSOCIATED LIPOCALIN: (NGAL)[(50)]

Involved in iron transport , released by neutrophils upon activation

Increased expression in several tissue in response to inflammation

Among the most up regulated genes in tubular cells after ischemic AKI in animal model.

Other markers:

Alanine aminopeptidase

Alkaline phosphatase

α-Glutathione -S- transferase

β2microglobulin

α 1microglobulin

Retinol binding protein

Microalbumin

Clusterin

Cysteine rich protein

Osteopontin , exosomal fetulin –A .

Haptogloin,haeme oxygenase,fibulin 1.

Vanin – 1- drug induced AKI

MANAGEMENT OF ACUTE KIDNEY INJURY

Prevention: [53]

In ischemic and nephrotoxic AKI ,there are no specific treatment modalities available, prevention is of paramount importance .Many patients with ischemic AKI can be prevented by attention to cardiovascular function and intravascular volume in high risk patients such as elderly and with preexisting CKD patients.

In case of ischemic AKI due to major surgery ,trauma, burns, cholera, aggressive restoration of intravascular volume decline the incidence of AKI.[43]

The incidence of nephrotoxic AKI can be declined by altering the dose and frequency of nephrotoxic drugs to body and glomerular filtration rate. Serum creatinine is an insensitive indicator of GFR and overestimates GFR in elderly and young patients. Adjusting drug dosage according to bioavailablity of drugs appear to decline kidney injury.[40]

The drugs such as diuretics, NSAIDS,ACE inhibitors, angiotensin receptor blockers,should be used caution with suspected true or effective hypovolemia or renovascular disease , they may precipitate ischemic AKI. Allopurinol and forced alkaline diuresis are useful prophylactic measures to prevent precipitation of uric acid crystals in renal tubules in patients at high risk for acute urate nephropathy (cancer chemotheraphy in haematological malignancy).

N acetyl cystine [43] limits acetaminophen induced kidney injury &should be given within 24 hours of ingestion.in radiocontrast nephropathy [45] hydration is an effective preventive approach ,volume expansion with bicarbonate containing IV fluids are superior to normal saline administration.

Specific treatment: [41]

Prerenal AKI is reversible upon correction of haemodynamic abnormality &post renal AKI resolves upon relief of obstruction.

PRERENAL AKI:

According to the type of fluid in hypovolemia the replacement fluid can be tailored. In case of moderate haemarrhage or plasma loss (burns or pancreatitis) isotonic saline can be used, in severe hypovolemia due to haemarrhage corrected with packed red blood cells. [67,68]

In case of urinary and gastrointestinal fluid loss hypotonic fluid are usually recommended , serum potassium &acid base must be monitored carefully and potassium,bicarbonate supplement should be given.

In case of cardiac failure Inotrophic agents, preload and afterload reducing agents ,mechanical ventilation such as intraaortic ballon pump.

In case of cirrhosis with ascitis fluid management is difficult, reversible AKI due true or effective hypovolemia must be distinguished from hepatorenal syndrome .

The contribution of hypovolemia due to AKI are assesed only by administration of fluid challenge tests[52]. Fluid administered slowly based on JVP, CVP and PCWP. Incase of refractory ascitis transjugular intrahepatic portosystemic shunt improve renal function by increased central volume and suppression of aldosterone and noradrenaline secration.

INTRINSIC AKI:

To attenuate injury or hasten recovery in ischemic & nephrotoxic AKI has been tried, some of them are ANP, low dose dopamine , endothelial antagonists , loop diuretic , calcium channel blockers, prostaglandin analogues, antioxidants, antibodies against leucocyte adhesion molecules, and IGF type1.

AKI due to other intrinsic renal disease such as acute glomeronephritis or vasculitis to steroids and immunosuppressive agents. Aggressive control of blood pressure is particularly important in malignant hypertension .

POSTRENAL AKI:

Obstruction of urethra or bladder neck is initially managed by transurethral or suprapubic bladder catheter.uretric obstruction managed initially by percutaneous cathetrisation of dilated renal pelvis or ureter.

PERITONEAL DIALYSIS : [54]

Peritoneal dialysis involves the transport of solutes and water across the membrane that seperates two fluid containing compartments- Blood in the peritoneal capillaries in renal failure contains excess of urea, creatinine,& other solutes: Dialysis solution in the peritoneal cavity which contains

sodium , chloride and lactate or bicarbonate & it is rendered hyperosmolar by the inclusion of a high concentration of glucose .

Physiology of peritoneal transport :

Diffusion : Uraemic solutes and potassium diffused from peritoneal capillary blood into the peritoneal dialysis solution where as glucose, lactate, calcium & bicarbonate in the opposite direction .

Diffusion depends upon

Concentration gradient.

Effective peritoneal surface area.

Molecular weight of solute concerned .

Ultrafiltration :

It results as a consequence of the osmotic gradient between the hypertonic dialysis solution and the relatively hypotonic peritoneal capillary blood .it depends upon

Concentration gradient for osmotic agent (eg:glucose).

Hydraulic conductance of peritoneal membranes .

Hydrostatic pressure gradient .

Oncotic pressure gradient.

Sieving.

Reflection coefficient for osmotic agents.

Effective peritoneal surface area.

Fluid absorption:

Intraperitoneal hydrostatic pressure

Effectiveness of lymphatics

Disadvantages of peritoneal dialysis:

Less efficient than haemoperfusion in treatment of acute kidney injury for flash pulmonary edema,poison or drug overdosage, acidosis ,hyperkalemia. Moderate protein could complicate mal nourished ,critically ill patients. However mortality due peritoneal dialysis and haemoperfusion are similar.

Contraindications : [55]

Absolute – recent surgery requiring abdominal drains

Pleuroperitoneal fistula

Relative contraindication – severe hypercatobolic state –decreased clearance

Abdominal wall cellulitis

Adynamic ileus

New aortic prosthesis

Dialysis solution:

Dextrose – 1.5grams/dl to 4.25 gms/dl

Osmolarity – 346mosm to 485 mosm

Ultrafiltration – 50-150 ml/exchange

Complication of peritoneal dialysis:

Peritonitis

Abdominal distension

Hypotension

Hypernatremia

Hyperglycemia

Hypoalbuminemia.

HAEMODIALYSIS: [56]

The blood is passed through an extracorporeal circulation where it is separated from the dialysis fluid by an artificial semipermeable membrane. water molecules and low molecular solutes in the two solution can pass through membrane pores and intermingle but large molecules such as proteins cannot pass.

Temporary access is established by the percutaneous insertion of catheter into a large vein (internal jugular vein, femoral , less commonly subclavian). venous catheters are commonly used for autocoagulation in case of acute kidney injury.

INSERTION LOCATION :

The optimal insertion site is right internal jugular vein. femoral vein cathetrization is a good choice when need for haemoperfusion is expected to less than one week. Subclavian site avoided - pneumothorax,subclavian artery perforation , brachial plexus injury.

COMPLICATIONS :

Immediate – arterial puncture

Pneumothorax

Haemothorax

Arrhythmias

Air embolism

Perforation of vein or cardiac chamber

Pericardial tamponade

Delayed -Thrombosis

Infection

Vascular strictureAV fistula

Injury to adjacent structure – brachial plexus injury Trachea Recurrent laryngeal nerve.

HAEMODIALYSIS PRESCRIPTION : [57,58]

Reduce the amount of dialysis for the initial one or two session , if predialysis blood urea nitrogen > 125 mg/dl dialysis session length reduced to two hour at a time & blood flow rate 250ml/min.

Longer initial dialysis session and increased blood flow rate results in disequilibrium syndrome, length of second session increased to 3 hours if the predialysis BUN < 100mg /dl . A typical 3 to 4 hours acute dialysis session will deliver a single pool Kt/v of only 0.9 with a equilibrated kt/v of 0.7 dialysate side urea removal may be even lower (evan et al -1999)

Data by schiffl et al -2002 found that mortality is reduced in patients with AKI dialysed six times per week as opposed to those receving every alternate day.

Dialyzer membrane:

Unsubsituted cellulose membrane had a lower pulmonary diffusion capacity- Heerero et al -2002

Subramanian et al 2006 – unsubsituted cellulose should not be used for acute dialysis

Dialysis solution (variable)

Base bicarbonate -145Mm

Potassium – 3.5mM

Calcium – 3.5mEq/L

Magnesium – 0.75mEq/L

Dextrose -200mg/dl

Phosphate –none

COMPLICATION DURING HAEMOPERFUSION:

Hypotension:

Volume related -high ultra filtration rate, Low dialysis sodium

Inadequate vasoconstriction – High dialysis solution temperature

Autonomic neuropathy

Antihypertensive medication

Eating during treatment

Anaemia

Acetate buffer

Cardiac factors – diastolic dysfunction ,

Arrhythmias

Ischemia

Myocardial infarction

Pericardial tamponade

Septicemia

Hemolysis

Air embolism

Muscle cramps –Hypotension , hypovolemia, high ultrafiltration rate , use of low sodium dialyzate solution.

Headache, Chest pain /back pain, Itching, Fevers /chills, Dialyzer reaction Arrhythmias, Cardiac tamponade, Intracranial bleeding Seizures, Haemolysis, Air embolism.

Visual & hearing loss – Evans et al found that transient blindness in patients with glaucoma and hearing loss due to endolymphatic hydrops.

Dialysis associated neutopenia & hypoxemia

Disequilibrium syndrome – systemic and neurologic symptoms associated with characteristic EEG abnormality.

Clinically- nausea, vomiting, restlessness , headache, seizures, obtundation & coma.

Etiology – acute increase in brain water content due to plasma solute level is rapidly lowered during dialysis , the plasma become hypotonic with respect to brain cells & water shift from plasma in to brain tissue.

Nutrition and life style changes in AKI patients:

Protein -1.2 g of proteins /kg daily-atleast 50 % of proteins had high biological value. Rocco et al in 2004 : 30 – 50 % of heamoperfusion patients intake of less than 1.0g/kg body weight per day.

Energy – less than 61 years -35 kcal/ kg/ day

More than 61 years 30 -35kcal /kg /day

In HEMO study intake based on dietary recall average is 23 – 27kcal /kg /day

Carbohydrates – 50 to 60 % of dietary intake which represents 1000 kcal or 250 grams of carbohydrate for a 2000 kcal diet.

Lipids – LDL cholesterol < 100 mg /dl

Fasting triglycerides - < 500 mg /dl

Therapeutic life style change :

Diet

Weight reduction

Increased physical activity

Abstinence from alcohol

Treat hypoglycemia.

MATERIALS & METHODS

INCLUSION CRITERIA:

All inpatients with clinical and / or biochemical evidence of acute kidney injury**.**

EXCLUSION CRITERIA:

- Patients with diabetes mellitus
- Patients with CKD
- Patients aged below 12 years.

METHODOLOGY:

Patients at risk of developing AKI (ie) history of preceding snake bite, ADD, febrile illness, septicemia, poisoning with symptoms suggestive of AKI were selected. Patients having one or more risk factors for CKD and patients already having CKD are excluded.

Patients social, demographic, economic and medical details were recorded in the proforma sheet. Also the history regarding the symptoms of AKI like decrease in urine output was recorded and the duration of symptoms (in hours) was also recorded. Base line clinical examination of patient was done. Vitals were recorded. Baseline investigations done. USG abdomen was done for all patients to assess the renal size and texture to rule

out CKD.other relevant investigation such as ANA,renal biopsy, fever profile was done.

The day the AKI criteria was fulfilled according to serum creatinine was noted as day -I. We used either the rise in serum creatinine of ≥ 0.3 mg/dl or ≥ 150 to 200 % from baseline to diagnose AKI.

Also the duration of stay in hospital is noted for all patients and the number of dialysis needed for those who developed AKI was also recorded.the outcome was compared with the cause, clinical and biochemical parameters.

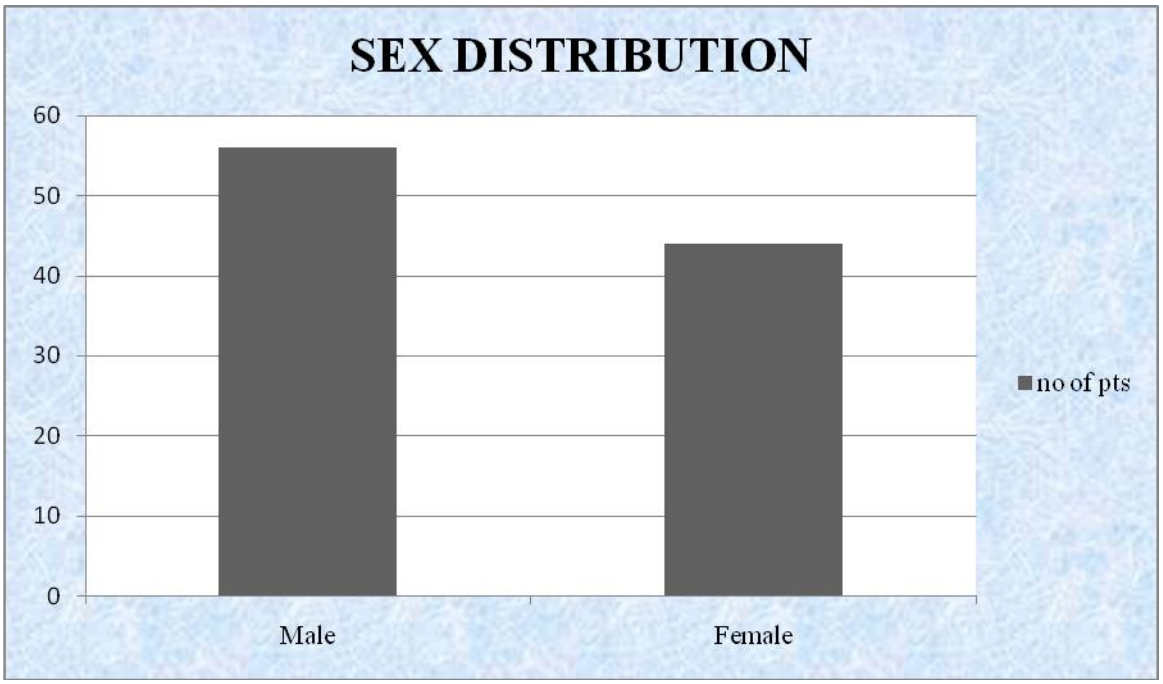
Statistical analysis

We calculated the mean ± standard deviations ($x \pm s$) or the medians for continuous variables and proportion of categorical variables. T-test was used to compare the continuous variables of normal distributions. Rank sum test was utilised for the continuous variables with abnormal distributions. Chi-square test was employed to compare categorical variable data. *P* value < 0.05 was considered significant. Data were analysed using SPSS11.5 software.

OBSERVATION & RESULTS

TABLE.1 GENDER DISTRIBUTION

SEX	No.of patients (n=100)	Percentage (%)
Male	56	56.0
Female	44	44.0

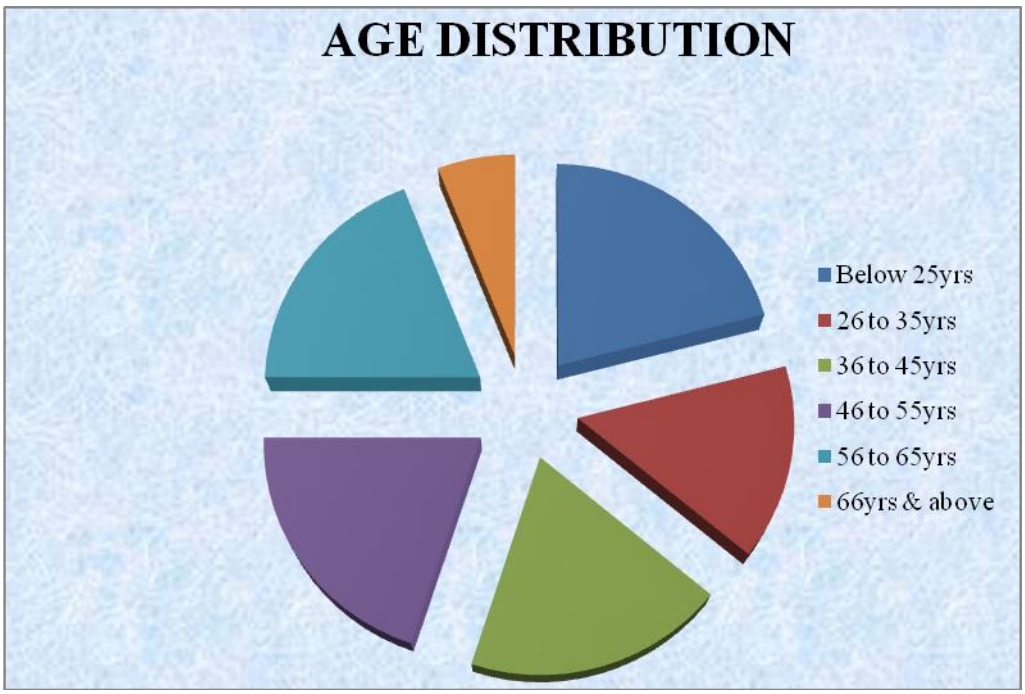


Males and female gender distribution in my study was almost equal with a slight predominance to male gender.

Sex difference doesn't show any significant difference in the outcome

TABLE.2 AGE DISTRIBUTION

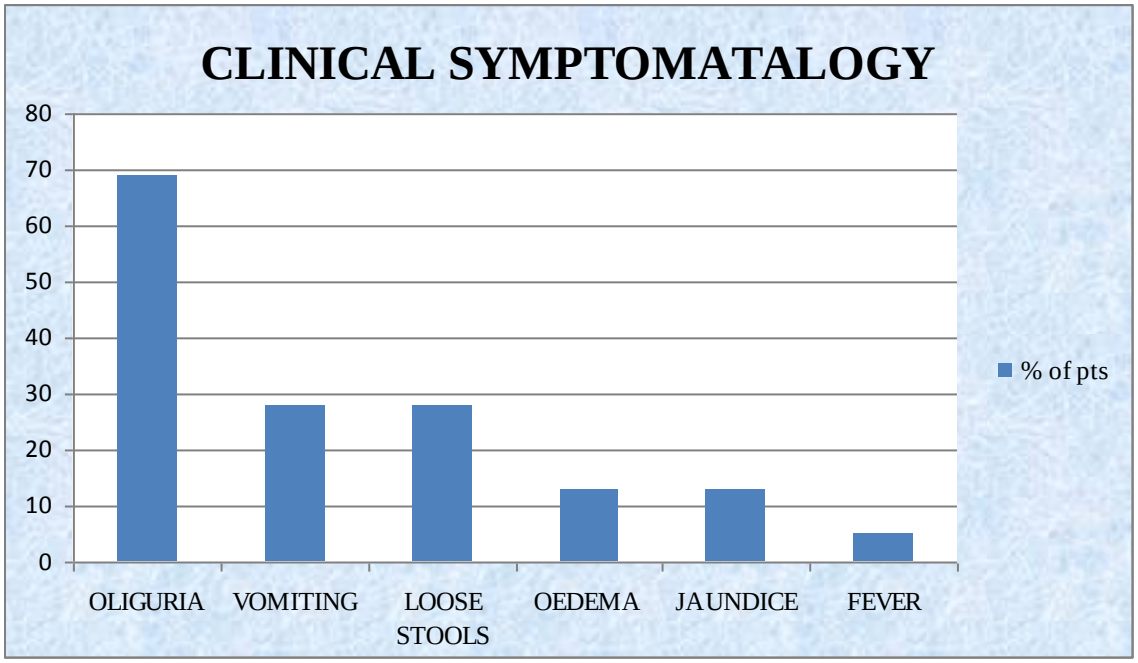
Age in years	No of patients (N=100)	Percentage %
Below 25yrs	21	21.0
26 to 35yrs	15	15.0
36 to 45yrs	19	19.0
46 to 55yrs	20	20.0
56 to 65yrs	19	19.0
66yrs & above	6	6.0



In our study group majority of them fall with in age limit of below 25 years even after excluding those below 12 yrs. Mean age group in my study was 42.6 years.

TABLE.3 CLINICAL SYMPTOMATALOGY

Signs &symptoms	No.of patients (n=100)	Percentage (%)
Oliguria	69	69.0
Vomiting	28	28.0
Loose stools	28	28.0
Oedema	13	13.0
Jaundice	13	13.0
Fever	05	05.0



This graphical representation shows varied clinical presentation of AKI. almost 70% of them had decreased urine output as a significant complaint.

In our part of the country loose stools (diarohea) also contributes to major part of AKI.

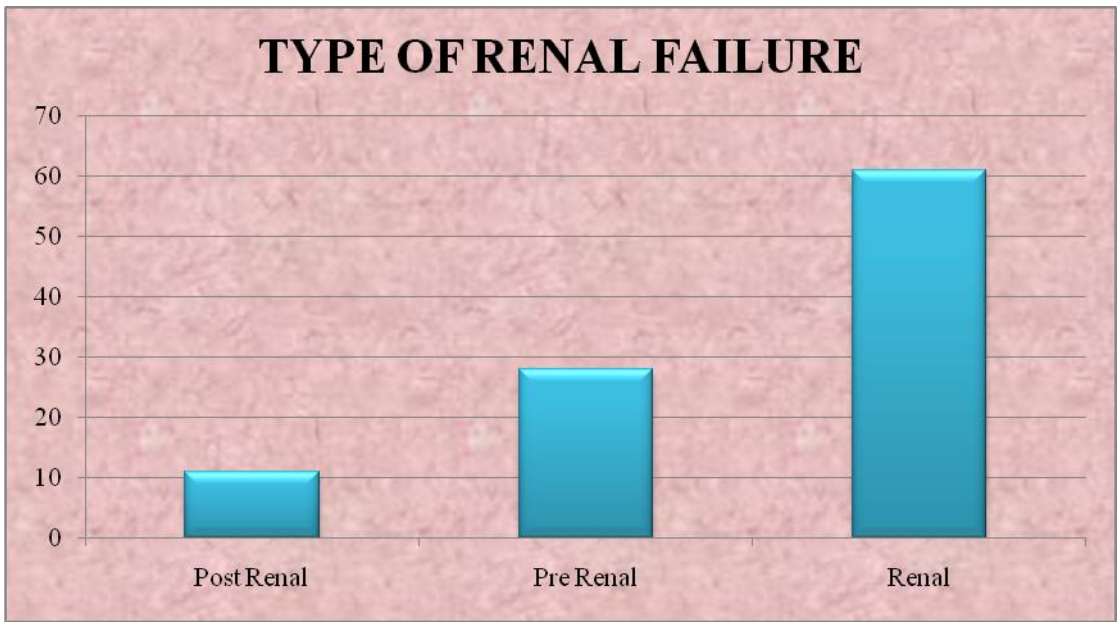
TABLE.4 CAUSES OF ACUTE KIDNEY INJURY

Diagnosis	No.of patients (n=100)	Percentage (%)
AGE	28	28.0
Bite	20	20.0
Septicemia	11	11.0
Mods	4	4.0
Fever	5	5.0
Poison	8	8.0
Glomerulonephritas	10	10.0
Drug induced	3	3.0
Boo/BPH	5	5.0
Ca cervix / Ca cervix RT	4	4.0
Renal calculi	2	2.0

This table represents various causes of acute kidney injury in our patients.

TABLE.5 TYPE OF RENAL FAILURE

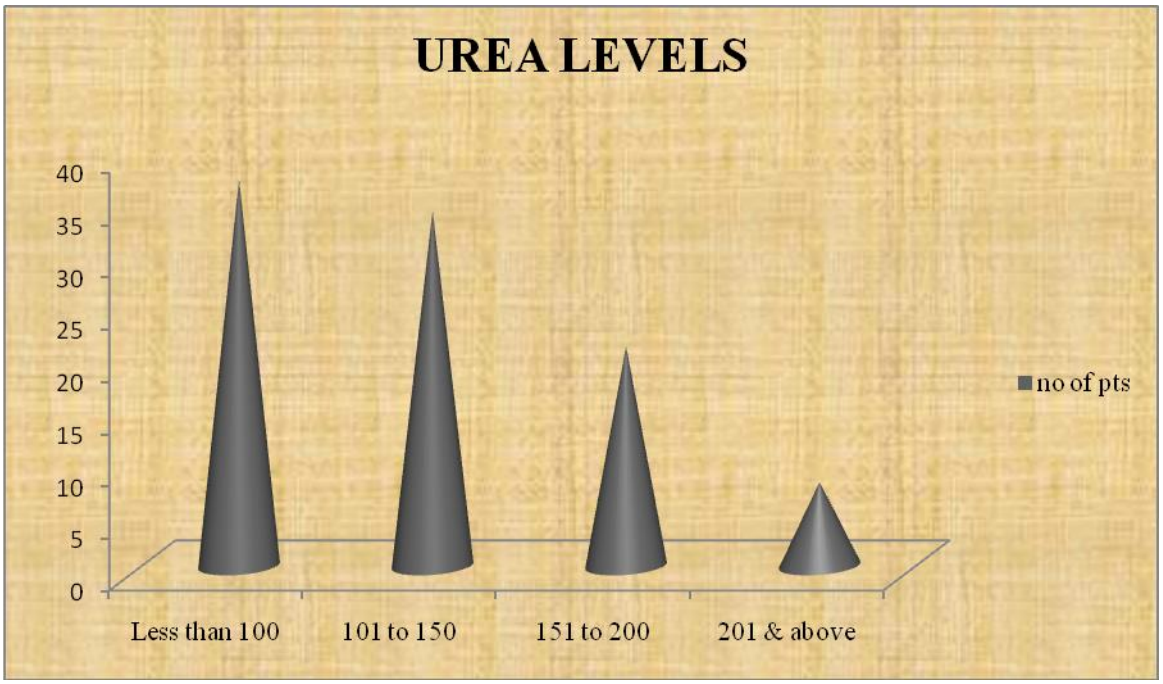
Type of renal failure	No.of patients (n=100)	Percentage (%)
Post Renal	11	11.0
Pre Renal	28	28.0
Renal	61	61.0



As mentioned in various literature in my study also intrinsic type of renal failure consititutes a major type of acute kidney injury.

TABLE.6 UREA LEVELS

UREA LEVELS	No.of patients (n=100)	Percentage (%)
Less than 100	37	37.0
101 to 150	34	34.0
151 to 200	21	21.0
201 & above	8	8.0



This is the graphical representation of severity of levels of urea in our patients.. Majority of them had urea levels between 101-150.

TABLE.7 SERUM CREATININE LEVELS

Serum creatinine (mg/dl)	No.of patients (n=100)	Percentage (%)
Less than 2	25	25.0
2 to 4	48	48.0
4 & above	27	27.0

Almost 50% of the individuals had creatinine levels between 2-4 mg/dl.

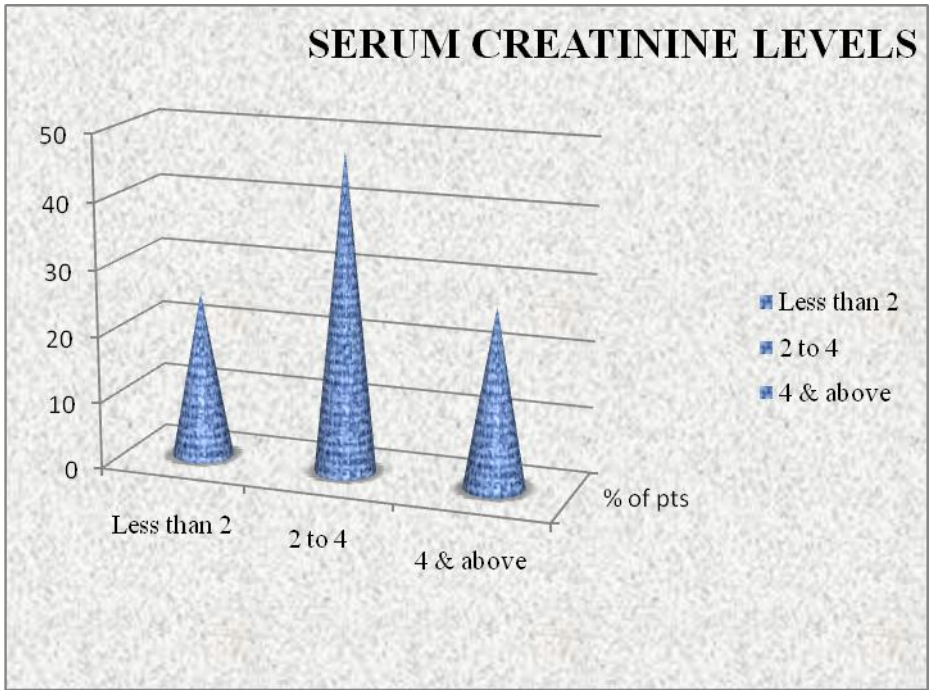


TABLE.8 URINE SODIUM LEVELS

URINE Na levels	No.of patients (n=100)	Percentage (%)
Less than 20	27	27.0
21 to 40	39	39.0
41 & above	34	34.0

Based on urine sodium levels 27% of individuals come under pre renal failure .

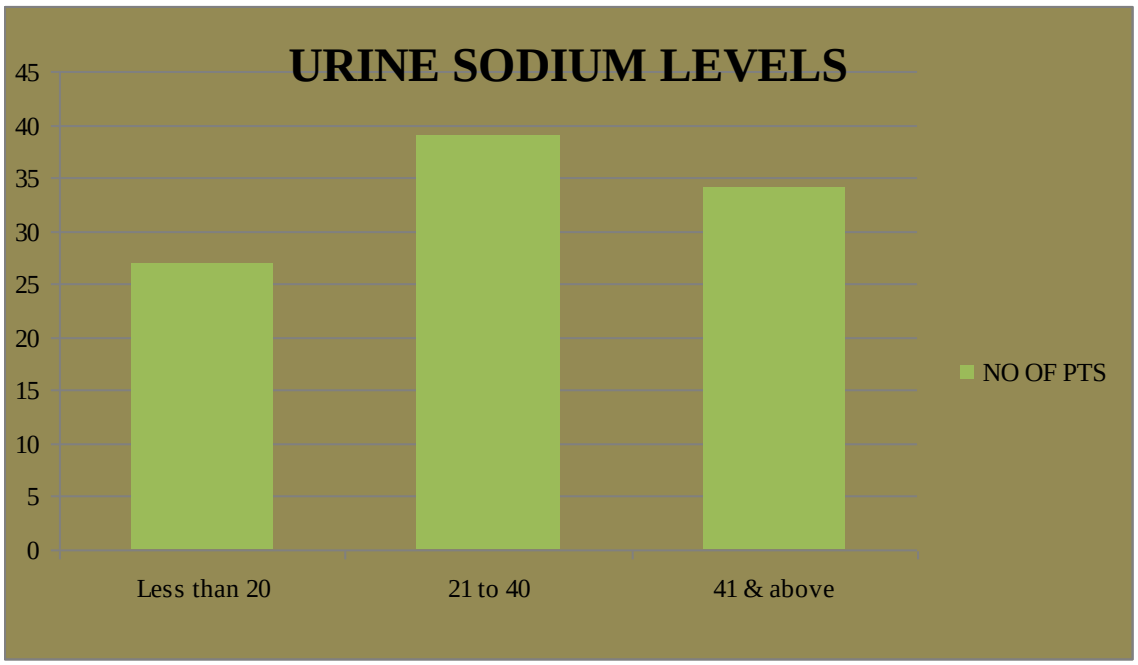


TABLE.9 SERUM POTASSIUM LEVELS

POTASSIUM meq/l	No.of patients	Percentage (%)
Less than 4.5	46	46.0
4.5 to 5.5	46	46.0
5.5 & above	8	8.0

Only 8% in our AKI population had hyperkalemia .

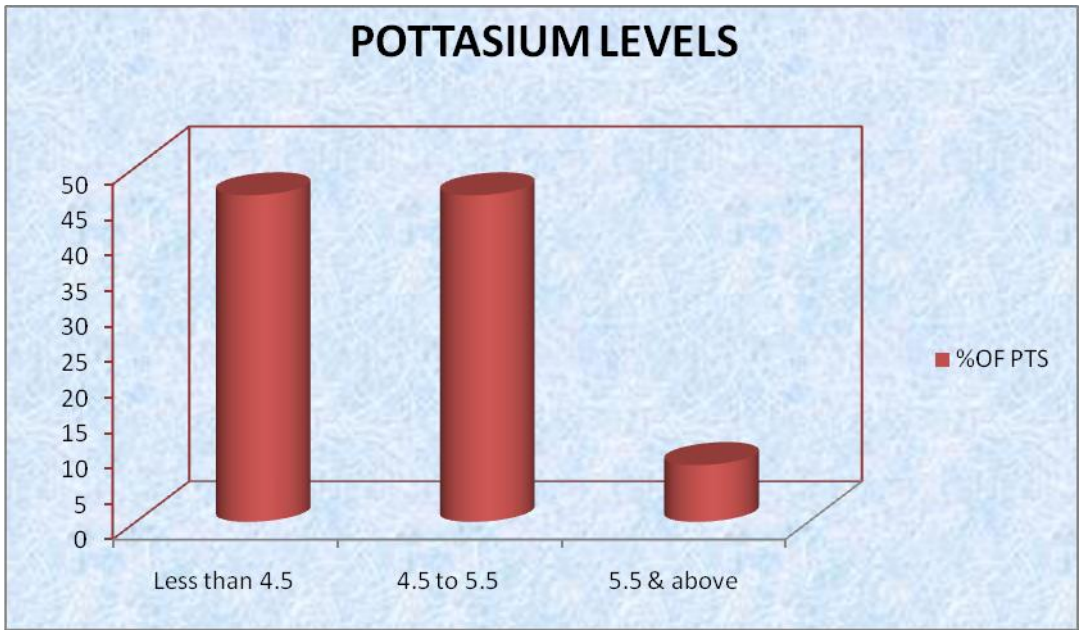
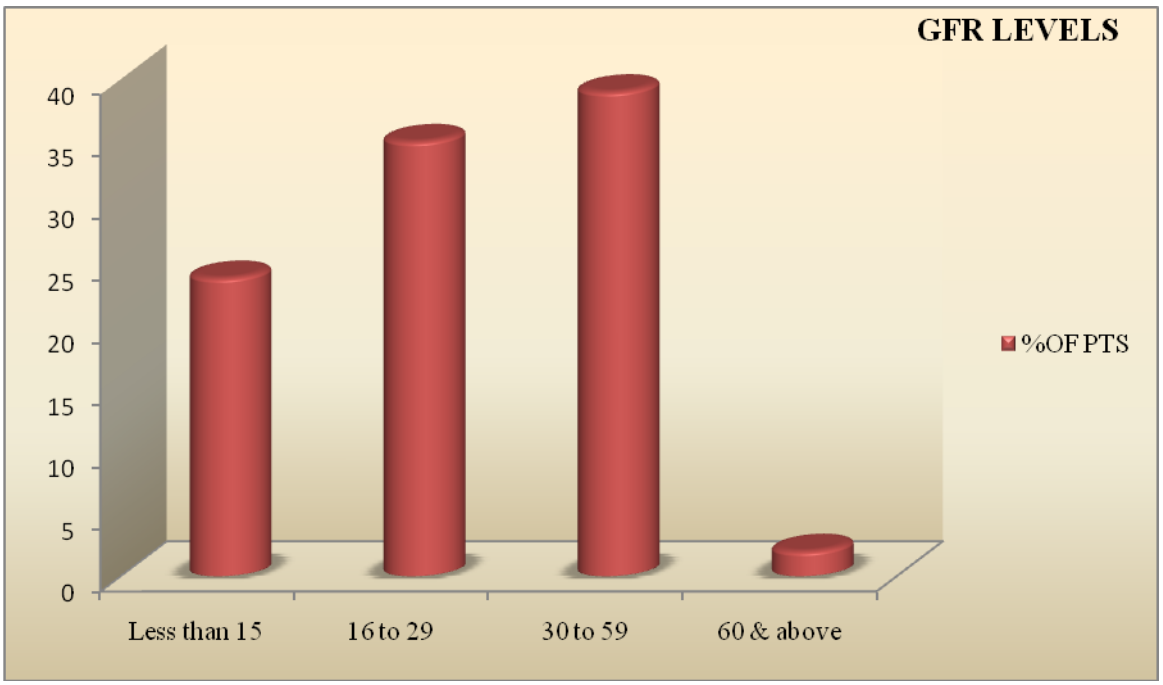


TABLE.10 GLOMERULAR FILTERATION RATE LEVELS

GFR ml/min	NO OF PATIENTS (n=100)	Percentage (%)
Less than 15	24	24.0
16 to 29	35	35.0
30 to 59	39	39.0
60 & above	2	2.0



Severity of renal failure based on gfr values are calculated and plotted in the above chart.

Almost 60% had gfr less than 30ml/min .

TABLE.11 TREATMENT MODALITIES

Treatment	No.of patients (n=100)	Percentage (%)
CONSERVATIVE	45	45.0
PERITONEAL DIALYSIS	30	30.0
HAEMODIALYSIS	26	26.0

This Chart Shows Percentage of Various Mode of Treatment In Acute Kidney Injury.

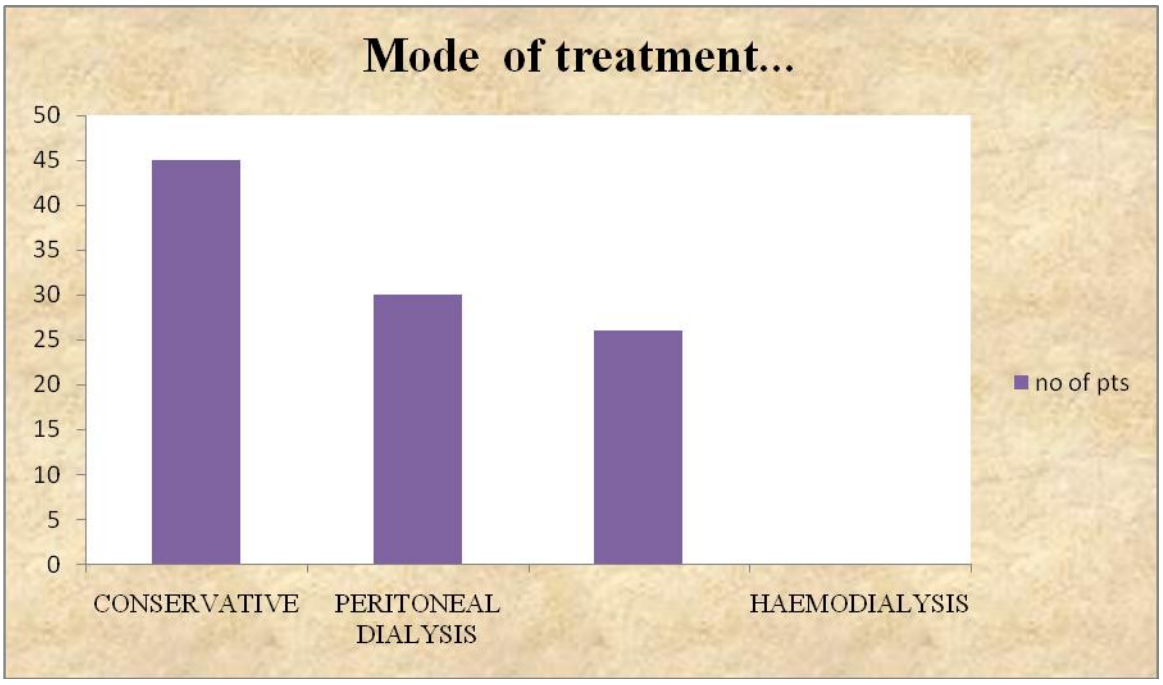
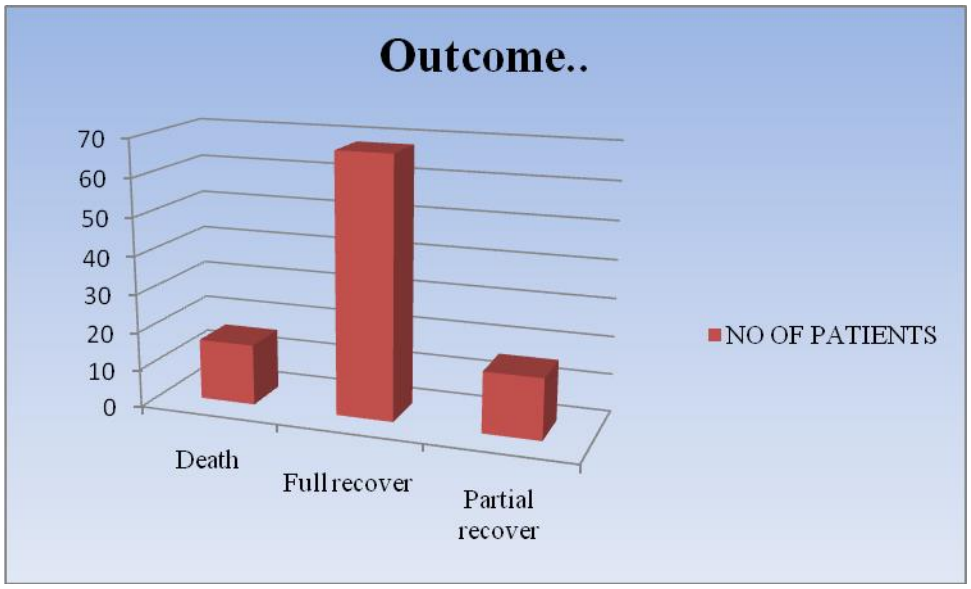


TABLE.12 OUTCOME

Outcome	No.of patients (n=100)	Percentage (%)
Death	16	16.0
Full recovery	68	68.0
Partial recovery	16	16.0



Almost 70% of our study population had complete recovery as evidenced by the creatinine values.but 16 % of them died.

TABLE.13 COMAPARISON OF UREA LEVELS WITH OUTCOME

	Death	Complete recovery	Partial recovery	Total	
Less than 100	1(6.3%)	31(45.6%)	5(31.3%)	37 (37%)	X^2=17.693 Df=6 .007<0.05 Significant
101 to 150	4(25%)	22(32.4%)	8(50%)	34 (34%)	
151 to 200	8(50%)	11(16.2%)	2(12.5%)	21 (21%)	
201 & above	3(18.8%)	4(5.9%)	1(6.3%)	8 (8%)	

The above table indicates there is association between urea levels with outcome, hence the calculated value is less than table value (p<.0.05).

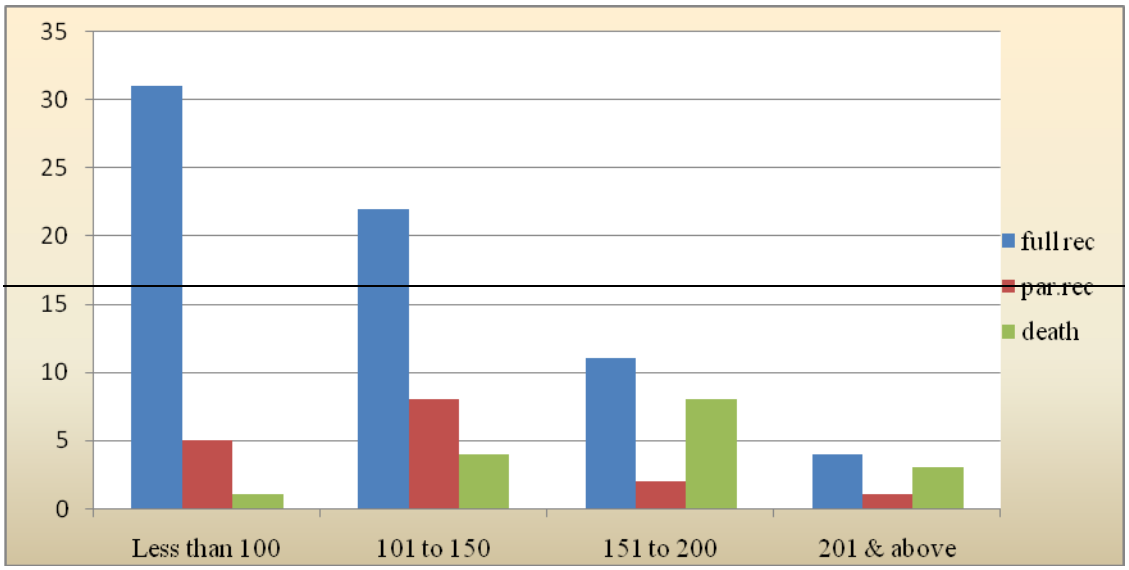


TABLE.14 COMARISON OF SERUM CREATININE LEVELS WITH OUTCOME

SERUM CREATININE	OUTCOME				Statistical inference
	Death	Complete recovery	Partial recovery	Total	
Less than 2	0	25(36.8%)	0	25 (25%)	X^2=62.980 Df=4 .000<0.05 Significant
2 to 4	0	38(55.9%)	10(62.5%)	48 (48%)	
4 & above	16(100%)	5(7.4%)	6(37.5%)	27 (27%)	

This table represents that there is associciation between creatinine and recovery status in our patients which is shown by statistically significant values.

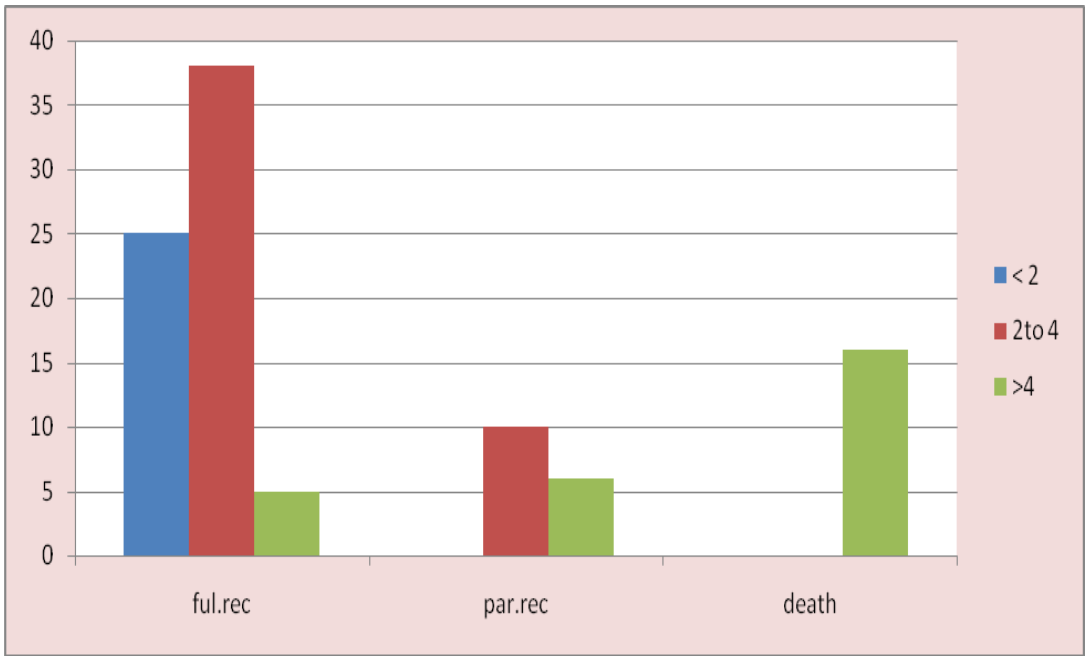


TABLE.15 COMPARISON OF UREA LEVELS WITH CONSERVATIVE TREATMENT

Urea	CONSERVATIVE TREATMENT			Statistical inference
	ABSENT	PRESENT	Total	
Less than 100	20(36.4%)	17(37.8%)	37(37%)	X^2=1.689 Df=3 .639>0.05 Not Significant
101 to 150	17(30.9%)	17(37.8%)	34(34%)	
151 to 200	12(21.8%)	9(20%)	21(21%)	
201 & above	6(10.9%)	2(4.4%)	8 (8%)	

Above table indicates that there is no association between urea and conservative treatment.hence the calculated values are greater than the table values where as the creatinine values correlated with the conservative line of treatment.

TABLE.16 COMPARISON OF SERUM CREATININE LEVELS WITH CONSERVATIVE TREATMENT

SERUM CREATININE	CONSERVATIVE TREATMENT			Statistical inference
	ABSENT	PRESENT	Total	
Less than 2	2(3.6%)	23(51.1%)	25 (25%)	X^2=35.411 Df=2 .000<0.05 Significant
2 to 4	29(52.7%)	19(42.2%)	48 (48%)	
4 & above	24(43.6%)	3(6.7%)	27 (27%)	

TABLE.17 COMPARISON OF UREA LEVELS WITH PERITONEAL DIALYSIS

UREA	PERITONEAL DIALYSIS			Statistical inference
	Done	Not done	Total	
Less than 100	16(53.3%)	21(30%)	37(37%)	X^2=5.711 Df=3 .127>0.05 Not Significant
101 to 150	7(23.3%)	27(38.6%)	34(34%)	
151 to 200	6(20%)	15(21.4%)	21(21%)	
201 & above	1(3.3%)	7(10%)	8 (8%)	

In compared with haemodialysis peritoneal dialysis does not show any correlation with elevated urea levels,where as elevated creatinine values correlates with peritoneal dialysis.

TABLE.18 COMAPARISON OF SERUM CRATININE WITH PERITONEAL DIALYSIS

SERUM CREATININE	PERITONEAL DIALYSIS			Statistical inference
	Done (n=30)	Not done (n=70)	Total (100%)	
Less than 2	2(6.7%)	23(32.9%)	25 (25%)	X^2=14.794 Df=2 .001<0.05 Significant
2 to 4	23(76.7%)	25(35.7%)	48 (48%)	
4 & above	5(16.7%)	22(31.4%)	27 (27%)	

TABLE.19 COMPARISON OF UREA LEVELS WITH HAEMODIALYSIS

UREA	HAEMODIALYSIS			Statistical inference
	Not done	Done	Total (100%)	
Less than 100	33(44.6%)	4(15.4%)	37 (37%)	X^2=9.937 Df=3 .019<0.05 Significant
101 to 150	25(33.8%)	9(34.6%)	34 (34%)	
151 to 200	12(16.2%)	9(34.6%)	21 (21%)	
201 & above	4(5.4%)	4(15.4%)	8 (8%)	

TABLE.20 COMPARISON OF SERUM CREATININE WITH HAEMODIALYSIS

SERUM CREATININE	**HAEMODIALYSIS**			**Statistical inference**
	Not done	**Done**	**Total**	
Less than 2	25(33.8%)	0	25 (25%)	X^2=52.464 Df=2 .000<0.05 Significant
2 to 4	43(58.1%)	5(19.2%)	48 (48%)	
4 & above	6(8.1%)	21(80.8%)	27 (27%)	

Above said tables indicate that there is a significant association in response to elevated urea,creatinine levels to haemodialysis as depicted by chi square test.

TABLE.21 COMAPARISON OF CONSERVATIVE TREATMENT WITH OUTCOME

CONSERVATIVE TREATMENT	OUTCOME				Statistical inference
	Death	Full recovery	Partial recovery	Total	
No	14(87.5%)	25(36.8%)	16(100%)	55 (55%)	X^2=29.055 Df=2 .000<0.05 Significant
Yes	2(12.5%)	43(63.2%)	0	45 (45%)	

TABLE.22 COMAPARISON OF PERITONEAL DIALYSIS WITH OUTCOME

PERITONEAL DIALYSIS	OUTCOME				Statistical inference
	Death	Full recovery	Partial recovery	Total	
Done	1(6.3%)	20(29.4%)	9(56.3%)	30 (30%)	X^2=9.559 Df=2 .008<0.05 Significant
Not done	15(93.8%)	48(70.6%)	7(43.8%)	70 (70%)	

The above tables illustrates that there is significant corelation between consevative treatment &peritoneal dialysis with outcome.

TABLE.23 COMPARISON OF HAEMODIALYSIS WITH OUTCOME

HAEMODIALYSIS	OUTCOME				Statistical inference
	Death	Full recovery	Partial recovery	Total	
Not done	1(6.3%)	63(92.6%)	10(62.5%)	74 (74%)	X^2=51.560 Df=2 .000<0.05 Significant
Done	15(93.8%)	5(7.4%)	6(37.3%)	26 (26%)	

The above table illustrates there is significant corelation between haemodialysis and outcome p <.0.05.

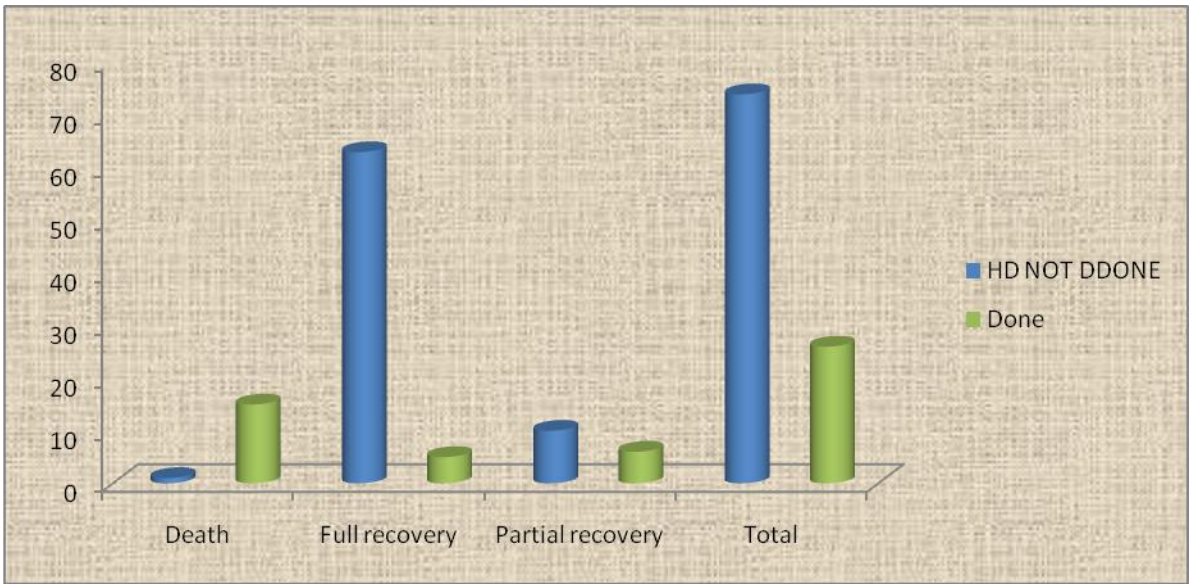
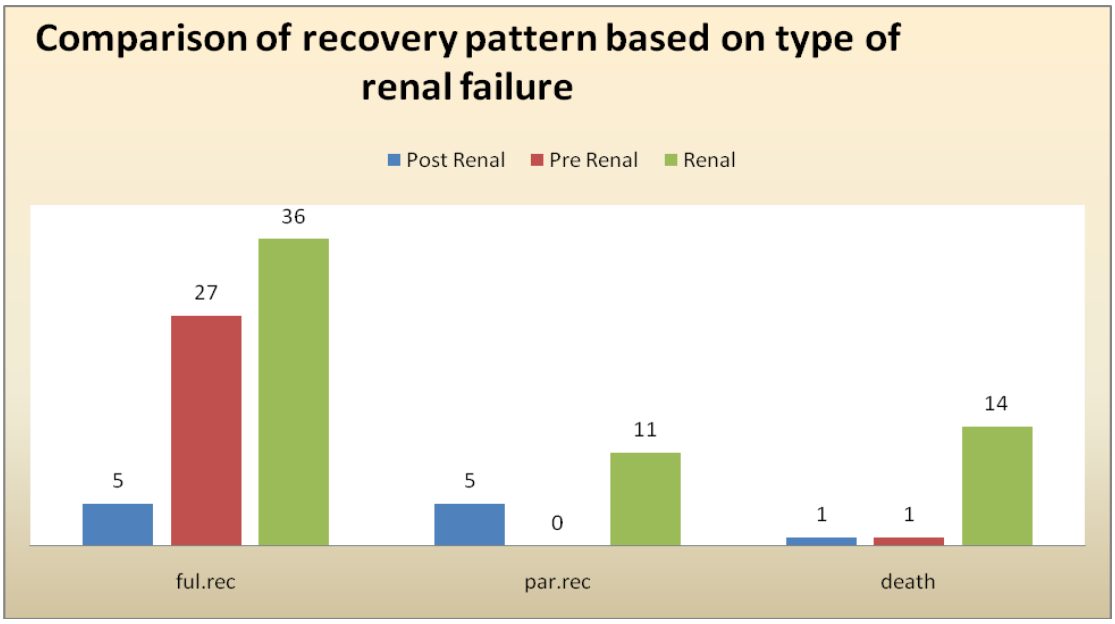


TABLE.24 COMPARISON OF RECOVERY BASED ON TYPE OF RENAL FAILURE

TYPE OF RENAL FAILURE	OUTCOME				Statistical inference
	Death	Complete recovery	Partial recovery	Total	
Post Renal	1(6.3%)	5(7.4%)	5(31.3%)	11 (11%)	X^2=20.349 Df=4 .019<0.05 Significant
Pre Renal	1(6.3%)	27(39.7%)	0	28 (28%)	
Renal	14(87.5%)	36(52.9%)	11(68.8%)	61 (61%)	

Both complete recovery and death is comparitively more in renal failure (intrinsic) as represented in the graph and table.



DISCUSSION

The present study is to determine the etiologies, characteristics and outcomes of acute kidney injury patients admitted to thanjavur medical college hospital from april 2013 to October 2013.

Demographic variation : Out of 100 patients included in the study 56 were males and 44 were females with a mean age of 42.6 years resulting in male :female ratio of 1:4 which is quite similar to 1.8:1 ratio of other developing countries .The M:F ratio in developed countries were 1:1.

Kandoth [70] showed that 67% of male & 23%of female patients are brought to the hospitals within 24 hours of oliguria. In our study youngest age of 13 years and oldest age of 80 years.

Berniech B et al.,[61] in which 58% were males &36%were females with mean age of 56.2 years.

Ravindra L. Mehta et al., in her study 59%males &41% were males with mean of 59.5years,in our study mean age was less compared to other two studies.

Study series	Males in %	Females in %	Mean age group
Present study	56%	44%	42.6
Bernieh B et al	58%	42%	56.2
Ravindra L et al	59%	41%	59.5

CLINICAL FEAUTURES:

A study of common signs & symptoms were made out. we noticed that oliguria and vomiting are most common presenting symptoms comprising of 69% and 28% respectively .the incidence of loose stools (28%), jaundice (13%) &fever (5%) were noticed.

This findings is compared with other studies done by Singhal AS et al., which showed that oliguria and vomiting was seen in 85.2% and 80% of patients respectively,

Liano Fet al ., [63] in her study found that hypotension was seen in 32.8% of patients . 52% of patients had hypotension in Bernieh et al study., and 20.6% of patients had hypotension in a study done by Singhal AS. In our study hypotension was seen in 12% of patients.

Signs &symptoms	Present study	Singhal AS et al [62]	Bernieh B et al	Liano F et al
Oliguria	68%	80.0%	78%	80%
Vomiting	28%	85.2%	80%	86%
Loose stools	28%	-	-	-
Oedema	13%	-	20%	-
Jaundice	13%	20%	-	-
Hypotension	12%	20.6%	52%	32.8%

CAUSES :

In our present study of 100 patients of AKI, about 28 patients had AKI due to prerenal cause ,most commonly acute gastroenteritis out of which 27 patients underwent conservative treatment with I.V fluids & other supportive measures.One patient underwent haemodialysis, 27 patients recovered completely and one patients died with a mortality of 6.3%.this was compared with study done by Liano F et al., which has shown prerenal AKI was seen in 21% of patients.

In the present study 61% of patients had AKI due to renal cause due to snake bite this is because the study was conducted in snake prevalent zone, other common etiologies are septicemia (11%), acute glomerulonephritis (10%), poison (8%), fever (5%), MODS(4%) ,drug induced (3%) respectively.

Etiology	Present study %	Singhal AS et al %	Bernieh et al %
AGE	28	12	9
BITE	20	20	-
SEPTICEMIA	11	12	58
POISON	8	-	-
FEVER	5	-	-
MODS	4	-	-
DRUG INDUCED	3	3	12
GLOMERULONEPHRITIS	10	-	-

In the present study, 11% due to post renal cause out of which 5 patients had complete recovery & 5 patients had partial recovery and one patient died.

OUTCOME:

The course of stay in hospital and outcome of these patients with acute kidney injury is variable.

In the present study out of 100 patients, 84 patients survived and about 16 patients expired. Among the survived 68 patients had complete recovery &16 patients had recovered partially. Most common cause of mortality are renal cause of AKI ,of this septicemia &MODS are predominant.

Out of 100 patients, 45 patients were treated conservatively ; 30 patients underwent peritoneal dialysis & 26 patients underwent haemodialysis.

There are about 28 patients with blood urea of more than 150mg/dl , out of which 11 patients had been treated conservatively and 17 patients with dialysis of which 12 died. There are about 27 patients with serum creatinine more than 4 meq/l , 3 patients had been treated conservatively ,24 patients with dialysis of which 16 died.

The overall mortality had been increase in patients with blood urea more than150mg/dl & serum creatinine more than 4meq/l.

Guerin et al., in his study found that incidence of AKI in hospitalized patients with serum creatinine more than 3.4meq/dl was 7.7% and need for renal replacement therapy is more. Overall hospital mortality due to AKI was 60% and 81% in patients developed AKI within one week of admission to ICU.

CONCLUSION

The present prospective observational study from thanjavur medical college ,thanjavur between april 2103 to October 2013 .

1. The clinical features were studied; it was observed that oliguria, vomiting are predominant symptoms in acute kidney injury.

2. Snake bite and acute diarrheal disease were the most common cause of AKI , however septicemia, MODS, poisoning, drugs were the other AKI cause. Leptospiral AKI was on the decline.

3. In snake bite, the ASV therapy time, bite to renal insufficiency time and coagulation abnormalities were the major prognostic factors predicting the final outcomes.

4. The mortality was 16 % with AKI admitted and 16 % of patients who survived had partial recovery at discharge, overall 55 % of patients required renal replacement therapy.

We observed that early diagnosis and early intervention are responsible for good survival rate in AKI.

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PROFORMA

Name: SI. NO:

Age: D.O.A:

Sex: D.O.D:

Occupation: I.P. NO:

Address:

Presenting Complaints:

1. History:

Oliguria	Yes	No	Vomiting/ nausea	Yes	no
Polyuria	Yes	No	Anorexia	Yes	No
Oedema	Yes	No	Rashes	Yes	No
Dyspnea	Yes	No	Pruritus	Yes	No
Cough	Yes	No	Jaundice	Yes	No
Weight loss	Yes	No	Fatigue	Yes	No
Loose stools	Yes	No	Haematuria	Yes	No
nephrotoxic drugs	Yes	No	Haemetemesis	Yes	No
Native treatment	Yes	No	Haemoptysis	Yes	No
Snake/ kathandu bite	Yes	No	Previous surgeries	Yes	No

2. Past History:

3. Family History:

4. Present medications:

5. Personal History:

Diet/ Appetite: Bowel/ Bladder habits:

Drug allergy:

6. Obstetric History (if significant):

7. General Physical Examination:

Consciousness

Built

Nutrition

Pallor / Icterus / Cyanosis / Skin rash

Signs of dehydration / JVP

Pedal Oedema / Lymph nodes / Conjunctival suffusion

Fundus

Other findings

Pulse rate: Blood pressure:

Respiratory rate: Temperature:

Weight: Height:

Peripheral Pulses

8. Systemic Examination:

ABDOMEN:

RESPIRATORY SYSTEM:

CARDIOVASCULAR SYSTEM:

CENTRAL NERVOUS SYSTEM:

9. CLINICAL DIAGNOSIS:

10. INVESTIGATIONS:

Date			
B.UREA(mg%)			
S.CREATININE(mg%)			
S.SODIUM(meq/l)			
S.POTASSIUM (meq/l)			

BLOOD:

FBS: CBC:

PPBS:

URINE SODIUM: URINE ROUTINE:

ECG –

CHEST X RAY -

USG Abdomen –

Right kidney - mm* mm. Echo- CMD-

Left kidney - mm* mm.Echo- CMD-

IMP:

SPECIAL INVESTIGATIONS (when indicated):

ANA / ds DNA
Anti GBM antibody
Rheumatoid Factor
Urine BJ protein
Serum EPP

11. TREATMENT GIVEN:

Conservative treatment : IVF / Antibiotics

Peritoneal Dialysis:

Haemodialysi:s

12. PATTERN OF RECOVERY:

Partial recovery / full recovery / renal biopsy

KEY TO MASTER CHART

M – Male

F - Female

WBCT – Whole Blood Clotting Time

PLT- Platelet Count

P- PRESENT

A-ABSENT

D- DONE

N-NOT DONE

F- Full Recovery

P-Partial Recovery

PD-Peritoneal Dialysis

HD- Hemodialysis

LFT- Liver Function Test.

CULTURE-Blood Culture

MP- Smear For Malarial Parasite

BT-Bleeding Time

CT-Clotting Time

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INTRODUCTION: Acute kidney injury (1) depicts the abrupt decline in renal function mostly occurs over the course (hours to days) and ends in retention of metabolic waste products and dysregulation of fluid, electrolytes, & acid base homeostasis. (1,2) During the past decades acute loss of kidney function previously referred to as acute renal failure has been modified to acute kidney injury with increased recognition of importance of relatively small changes in renal function on both short & long term clinical outcomes. The kidneys being relatively unique among other organs of the body in its ability to recover from almost complete loss of function, AKI may develop in a wide variety of...

S.NO	IP.No	AGE	SEX	OLIGURIA	EDEMA	diarrhea	hypotension	vomitting	jaundice	POISON	BITE	DRUG	FEVER	UREA	creatinine	GFR	SODIUM	potassium	urine sodium	relevant investigation	type of renal failure	diagnosis	conservative treatment	PD	HD	OUTCOME
1	1467514	69	M	P	A	A	A	p	P	A	P	A	A	126	4.8	13	139	4.9	46	WBCT,PLT	renal	snake bite	0	N	p	D
2	1467472	60	M	P	A	A	A	p	p	A	A	P	A	125	3.8	18	149	4.2	51		renal	drug induced	0	D	N	F
3	1467595	50	M	P	A	A	A	p	P	A	P	A	A	142	4.3	16	136	5.1	60	WBCT,PLT	renal	snake bite	0	N	p	F
4	1468726	35	M	P	A	P	p	A	A	A	A	A	A	224	2.4	33	150	4.8	14		pre-renal	AGE	0	N	N	F
5	1469929	60	M	p	A	A	A	A	A	A	A	A	A	158	3.8	18	137	5.2	47	usg abd	postrenal	BPH	0	D	N	F
6	1471079	55	M	p	A	A	A	A	A	A	A	A	A	108	2.8	26	151	4.8	36	usg abd	postrenal	BOO	0	D	N	p
7	1471106	40	M	P	p	A	p	p	A	A	A	A	A	160	4.4	16	138	5.2	38	culture	renal	septicemia	0	N	p	p
8	1482385	44	M	P	A	A	A	A	A	A	A	P	A	96	3.5	21	131	4	49		renal	drug induced	0	D	N	F
9	1443604	40	F	A	A	A	A	A	p	A	A	A	A	75	4.9	11	152	5.4	56	culture	renal	septicemia	0	N	p	D
10	1447556	50	F	P	A	A	A	A	P	A	P	A	A	132	5.2	13	139	5.1	48	WBCT,PLT	renal	snake bite	1	N	N	F
11	1451466	50	F	P	A	P	A	A	A	A	A	A	A	142	1.9	30	145	4.3	21		pre-renal	AGE	1	N	N	F
12	1460236	60	F	P	A	A	A	A	A	A	P	A	A	65	2.1	26	137	4.6	28	WBCT,PLT	renal	snake bite	1	N	N	F
13	1462632	65	F	p	A	A	A	p	A	A	A	A	A	204	4.9	10	144	5.8	35	usg abd	postrenal	ca cervix	0	N	p	p
14	1474702	57	F	P	A	A	A	A	A	A	P	A	A	174	5.9	8	136	4.9	29	WBCT,PLT	renal	snake bite	0	D	N	F
15	1474760	33	F	P	A	P	p	A	A	A	A	A	A	215	2.2	28	137	3.8	18		pre-renal	AGE	1	N	N	F
16	1455784	35	M	A	A	A	A	p	A	A	A	A	A	158	5.8	12	124	6.2	86	culture	renal	septicemia	0	N	p	D
17	1476060	60	F	P	A	A	A	p	A	A	A	P	A	68	2.3	23	139	4.6	36		renal	drug induced	1	N	N	F
18	1411358	45	F	p	p	A	A	p	A	A	A	A	A	148	5.2	10	134	4.8	42	usg abd	postrenal	ca cervix	0	D	N	p
19	1412569	18	M	P	A	P	A	A	A	A	A	A		198	2.3	45	124	4.1	19		pre-renal	AGE	1	D	N	F
20	1411939	35	F	P	A	P	A	A	A	A	A	A	A	298	3.9	14	129	5.2	14		pre-renal	AGE	0	D	N	F
21	1412884	16	M	A	A	A	A	A	A	A	A	A	A	68	2.2	43	136	4.6	29	biopsy	renal	PSGN	0	D	N	p
22	1414766	17	F	A	A	A	A	A	A	A	A	A	A	95	3.5	19	138	4.9	32	biopsy	renal	PSGN	0	D	N	F
23	1417298	32	F	P	A	P	A	A	A	A	A	A	A	185	1.9	33	142	4.5	18		pre-renal	AGE	1	N	N	F
24	1420973	24	M	A	p	A	A	A	A	A	A	A	A	136	4.8	16	139	5.8	36	biopsy	renal	RPGN	0	N	p	D
25	1422462	17	M	A	A	A	A	A	A	A	A	A	A	64	2.1	45	138	4.3	26	biopsy	renal	PSGN	1	N	N	F
26	1425073	19	M	P	A	P	A	A	A	A	A	A	A	186	2.8	32	141	4.9	15		pre-renal	AGE	1	N	N	F
27	1427679	45	F	A	p	A	p	p	A	A	A	A	A	182	6.9	7	139	5.2	39	culture	renal	septicemia	0	N	p	D
28	1427577	42	F	P	A	A	A	p	P	A	P	A	A	61	1.9	32	138	4.8	32	WBCT,PLT	renal	snake bite	1	N	N	F

S.NO	IP.No	AGE	SEX	OLIGURIA	EDEMA	diarrhea	hypotension	vomitting	jaundice	POISON	BITE	DRUG	FEVER	UREA	creatinine	GFR	SODIUM	potassium	urine sodium	relevant investigation	type of renal failure	diagnosis	conservative treatment	PD	HD	OUTCOME
29	1429298	33	M	A	A	A	p	p	P	A	A	A	A	132	4.8	15	124	5.3	39	LFT,ABG	renal	mods	0	N	p	D
30	1428935	12	M	P	A	P	A	A	A	A	A	A	A	108	1.6	66	136	4.1	16		pre-renal	AGE	1	N	N	F
31	1430366	50	F	A	A	A	p	p	p	A	A	A	A	132	4.6	11	129	3.6	39	culture	renal	septicemia	0	N	p	p
32	1430238	40	F	A	A	A	A	A	A	A	A	A	A	152	5.2	12	112	3.1	42	LFT,ABG	renal	mods	0	N	p	D
33	1430324	70	M	P	A	A	A	A	A	A	P	A	A	64	2.2	32	139	3.8	45		renal	kathandu	1	N	N	F
34	1435301	18	M	P	A	A	A	p	p	A	A	A	P	62	2.4	37	142	4.4	28	mp,lepto	renal	malaria	1	N	N	F
35	1437953	22	M	P	A	P	A	A	A	A	A	A	A	101	2.1	37	135	4.6	18		pre-renal	AGE	1	N	N	F
36	1441343	14	F	P	A	A	A	A	A	A	P	A	A	68	1.9	39	142	3.9	36	WBCT,PLT	renal	snake bite	1	N	N	F
37	1442043	40	F	A	A	A	p	p	p	A	A	A	A	157	5.8	9	108	2.1	104	LFT,ABG	renal	mods	1	N	p	D
38	1442165	33	M	A	A	A	A	A	A	A	A	A	A	132	4.4	17	145	5.2	53	biopsy	renal	RPGN	0	N	p	F
39	1443749	13	M	P	A	P	A	A	A	A	A	A	A	194	2.1	47	138	4.6	14		pre-renal	AGE	1	N	N	F
40	1445364	56	F	P	A	A	A	A	A	A	P	A	A	53	1.7	34	135	4.1	49	WBCT,PLT	renal	snake bite	1	N	N	F
41	1446661	37	F	p	p	A	A	A	A	A	A	A	A	92	2.9	20	141	3.6	39	biopsy	renal	RPGN	0	D	N	p
42	1447724	34	M	P	A	A	A	A	A	A	P	A	A	96	3.6	21	139	4.1	26	WBCT,PLT	renal	snake bite	0	N	p	F
43	1448164	65	F	A	A	A	P	A	A	A	A	A	A	150	5.2	9	144	5.6	106	culture	renal	septicemia	0	D	N	p
44	1447694	38	F	A	A	A	A	A	A	A	A	A	A	102	3.8	15	139	4.8	101	biopsy	renal	FSGS	0	N	p	p
45	1451129	23	M	P	A	A	A	P	p	A	A	A	P	65	2.2	40	135	4.1	23	mp,lepto	renal	leptosp	1	N	N	F
46	1454277	20	F	P	A	P	A	A	A	A	A	A	A	138	1.6	44	139	4.2	16		pre-renal	AGE	1	N	N	F
47	1455970	68	F	A	A	A	p	p	p	A	A	A	A	186	6.2	8	126	5.9	95	lFT,ABG	renal	mods	1	N	p	D
48	1458290	68	M	P	A	A	A	A	A	A	P	A	A	62	1.8	41	135	4.1	32	WBCT,PLT	renal	snake bite	1	N	N	F
49	1458912	55	M	p	A	A	A	A	A	A	A	A	A	156	3.2	22	142	4.8	37	usg abd	postrenal	renal calculi	0	D	N	F
50	1460477	34	M	P	A	P	A	A	A	A	A	A	A	139	1.9	45	139	4.9	19		pre-renal	AGE	1	N	N	F
51	1463208	65	F	p	A	A	A	A	A	A	A	A	A	109	2.2	24	141	4.6	34	usg abd	postrenal	BPH	1	N	N	F
52	1457479	25	M	P	A	A	A	A	A	A	P	A	A	72	2.8	30	138	4.1	42	WBCT,PLT	renal	snake bite	0	D	N	F
53	1481607	57	M	P	A	P	A	A	A	A	A	A	A	101	1.1	74	135	3.9	16		pre-renal	AGE	1	N	N	F
54	1481497	65	M	P	A	A	A	A	A	A	P	A	A	98	3.2	21	141	4.8	46	WBCT,PLT	renal	snake bite	0	N	p	F
55	1435071	53	M	p	A	A	A	A	A	A	A	A	A	109	2.6	28	134	4.9	52	usg abd	postrenal	renal calculi	0	D	N	F
56	1463918	60	F	P	A	P	A	A	A	A	A	A	A	162	1.8	31	146	3.7	17		pre-renal	AGE	1	N	N	F

S.NO	IP.No	AGE	SEX	OLIGURIA	EDEMA	diarrhea	hypotension	vomitting	jaundice	POISON	BITE	DRUG	FEVER	UREA	creatinine	GFR	SODIUM	potassium	urine sodium	relevant investigation	type of renal failure	diagnosis	conservative treatment	PD	HD	OUTCOME
57	1472245	42	F	A	p	A	A	p	A	A	A	A	A	154	5.3	10	134	4.8	52	culture	renal	septicemia	0	N	p	p
58	1473098	52	M	P	A	A	A	p	A	A	P	A	A	66	2.1	36	144	4.2	32		renal	bee sting	1	N	N	F
59	1475610	43	F	P	A	P	A	A	A	A	A	A	A	145	1.8	33	136	3.6	13		pre-renal	AGE	1	N	N	F
60	1476937	12	F	A	A	A	A	A	A	A	A	A	A	74	2.2	34	145	3.8	53	biopsy	renal	PSGN	0	D	N	F
61	1476063	48	F	A	A	A	A	A	A	A	A	A	A	97	3.2	17	139	4.9	65	biopsy	renal	FSGS	0	N	p	F
62	1475504	46	M	P	A	P	A	A	A	A	A	A	A	176	1.9	41	138	4.8	19		pre-renal	AGE	1	N	N	F
63	1477261	43	F	A	p	A	A	A	A	A	A	A	A	184	6.2	8	121	5.6	41	culture	renal	septicemia	0	N	p	D
64	1476252	50	F	p	p	A	A	A	A	A	A	A	A	185	4.2	12	146	4.9	37	usg abd	postrenal	ca cervix	0	D	N	D
65	1479495	26	M	P	A	P	A	A	A	A	A	A	A	142	1.6	56	138	4.8	16		pre-renal	AGE	1	N	N	F
66	1478260	31	M	P	A	P	A	A	A	A	A	A	A	164	1.8	48	142	3.9	12		pre-renal	AGE	1	N	N	F
67	1464299	65	F	P	A	A	A	A	A	A	P	A	A	64	2.2	19	143	4.6	38	WBCT,PLT	renal	snake bite	1	N	N	F
68	1464262	36	M	A	A	A	A	p	A	A	A	A	A	60	2	41	146	4.2	35	LFT,ABG	renal	Parquat pois	0	D	N	F
69	1466254	55	FF	P	A	A	A	A	A	A	P	A	A	59	2.3	24	134	4.8	45	WBCT,PLT	renal	snake bite	0	D	N	F
70	1466258	55	F	P	A	P	p	A	A	A	A	A	A	200	2	28	138	3.9	16		pre-renal	AGE	0	D	N	F
71	1469800	55	M	p	A	A	A	A	A	A	A	A	A	130	2.1	26	146	4.4	39	usg abd	postrenal	BPH	0	N	N	p
72	1470603	24	F	A	A	A	A	p	A	A	A	A	A	180	6	10	126	5.3	42	CPK	renal	vasmol pois	0	N	p	D
73	1470644	45	M	A	p	A	A	p	A	A	A	A	A	211	7.3	9	128	6.2	36	culture	renal	septicemia	0	N	p	D
74	1469543	42	M	A	A	A	p	p	A	A	A	A	A	90	3	25	143	4.9	32	BT,CT,LFT	renal	ratol poison	0	D	N	p
75	1472694	48	F	P	A	A	A	A	A	A	P	A	A	88	2.8	20	137	4.4	56	WBCT,PLT	renal	snake bite	1	N	N	F
76	1471731	55	M	P	A	P	A	A	A	A	A	A	A	222	2.3	32	142	3.8	16		pre-renal	AGE	1	N	N	F
77	1473979	12	M	P	A	P	A	A	A	A	A	A	A	150	1.8	58	139	3.4	20		pre-renal	AGE	1	N	N	F
78	147438	18	M	A	A	A	A	A	A	A	A	A	A	80	2.6	35	138	4.1	65	biopsy	renal	PSGN	0	D	N	p
79	1479275	35	F	P	A	P	A	A	A	A	A	A	A	120	1.9	32	143	4.9	17		pre-renal	AGE	1	N	N	F
80	1481520	15	M	P	p	A	A	p	p	A	A	A	P	92	3.6	25	145	4.3	39	mp,lepto	renal	malaria	0	D	N	F
81	1483222	33	M	P	A	P	A	A	A	A	A	A	A	146	1.8	47	128	5.1	14		pre-renal	AGE	1	N	N	F
82	1468233	22	M	P	A	A	A	A	A	A	P	A	A	84	2.6	33	139	3.4	29	WBCT,PLT	renal	snake bite	1	N	N	F
83	1469565	46	M	A	A	A	A	A	A	A	A	A	A	111	3.7	19	143	5.5	64		renal	Parquat pois	0	N	p	p
84	1468004	65	M	P	A	P	A	A	A	A	A	A	A	123	1.9	39	136	3.9	15		pre-renal	AGE	1	N	N	F

S.NO	IP.No	AGE	SEX	OLIGURIA	EDEMA	diarrhea	hypotension	vomitting	jaundice	POISON	BITE	DRUG	FEVER	UREA	creatinine	GFR	SODIUM	potassium	urine sodium	relevant investigation	type of renal failure	diagnosis	conservative treatment	PD	HD	OUTCOME
85	1469896	60	F	p	p	A	A	p	A	A	A	A	A	104	2.1	26	142	3.5	39	usg abd	postrenal	ca cervix,RT	0	D	N	p
86	1467387	60	F	P	A	P	A	A	A	A	A	A	A	111	1.8	31	141	3.6	12		pre-renal	AGE	1	N	N	F
87	1469161	40	M	A	A	A	A	A	A	A	A	A	A	222	7.2	10	152	5.1	42	cpk	renal	vasmol pois	0	N	p	D
88	1469862	50	M	A	p	A	A	p	A	A	A	A	A	96	3.5	20	139	4.9	555	culture	renal	septicemia	0	D	N	p
89	1469946	58	M	P	A	P	A	A	A	A	A	A	A	130	1.7	45	137	3.8	18		pre-renal	AGE	1	N	N	F
90	1464738	72	M	p	A	A	A	A	A	A	A	A	A	56	1.8	40	136	4.2	16	usg abd	postrenal	BPH	1	N	N	F
91	1470171	50	M	P	A	A	A	A	A	A	P	A	A	92	3.1	23	152	5.4	37	WBCT,PLT	renal	snake bite	0	D	N	F
92	1468512	32	M	A	A	A	A	A	A	A	A	A	A	89	2.6	31	139	4.6	33		renal	cu so4 pois	0	D	N	F
93	1478963	65	M	P	A	A	A	A	A	A	P	A	A	49	1.9	40	138	3.8	62	WBCT,PLT	renal	snake bite	1	N	N	F
94	1473698	45	F	P	A	P	A	A	A	A	A	A	A	146	2.8	27	146	3.6	21		pre-renal	AGE	1	N	N	F
95	1472589	26	M	P	A	A	A	A	A	A	A	A	P	59	3.1	27	143	4.7	36	MP,lepto	renal	puo	0	D	N	F
96	1472356	12	F	A	A	A	A	A	A	A	A	A	A	95	4.1	17	137	5.3	41		renal	unknown poi	0	D	N	F
97	1482563	47	M	A	p	A	A	p	A	A	A	A	A	261	8.4	8	123	5.9	54	culture	renal	septicemia	0	N	p	D
98	1472369	80	F	P	A	P	p	p	A	A	A	A	A	149	4.6	10	135	4.1	17		pre-renal	AGE	0	N	p	D
99	1472589	39	M	A	A	A	A	p	A	A	A	A	A	121	3.5	21	141	3.9	37		renal	cu so4 pois	0	D	N	F
100	1487966	62	F	P	A	A	A	A	A	A	A	A	P	69	2.1	26	138	3.8	34	MP,lepto	renal	puo	1	N	N	F